JUNE,
1939

The JOURNAL of SPEECH DISORDERS

Editor

G. OSCAR RUSSELL, PH. D.
Columbus, Ohio

Collaborating Editors

JEAN TARNEAUD, M. D.
Paris, France

DOUGLAS GUTHRIE, M. D.
Edinburgh, Scotland

M. NADOLECZNY, M. D.
München, Germany

H. BERING LIISBERG, Ph.D.
Copenhagen, Denmark

YOSHIO HAYASHI, PH. D.
Tokyo, Japan

F. HOGEWIND, M. D.
Den Haag, Holland

M. SEEMANN, M. D.
University of Prague
Czechoslovakia

ARNALDO MALAN, M. D.
Corso Oporto, 40
Torina, Italy

Official Publication, of the American Speech Correction Association in its 14th year.
Editorial Office: Speech Clinic, Ohio State University, Columbus, Ohio.
Volume 4 : Number 2

UNIVERSITY OF MICHIGAN
THE HORACE H. RACKHAM
SCHOOL OF GRADUATE STUDIES
ANN ARBOR
INSTITUTE FOR HUMAN ADJUSTMENT

$3.00 per Year
Single issue $1.00

CONTENTS

PAGE

Practical Aspects of Speech Correction,
Max A. Goldstein, M. D., LL. D. 99

Palatography and Speech Improvement,
Elbert R. Moses, Jr., Ph. D. 103

The Historical Background of the Modern Speech Clinic,
G. M. Klingbeil 115

Respiratory Cardiac Arrhythmia in Stuttering,
Martin F. Palmer, M.A., Sc.D.; Anna Mae Gillett, A.B. 133

Ear Training in the Treatment of Articulation Disorders,
C. Van Riper 141

Studies in the Psychology of Stuttering, XI. Stuttering in Relation to Position in the Family.....*Julian B. Rotter* 143

Studies in the Psychology of Stuttering, XII. Emphatic Breathing of Auditors While Listening to Stuttering Speech.....*Stanley Ainsworth* 149

Elongation of the Cleft Palate to Gain Better Nasopharyngeal Closure.....*James Barrett Brown, M.D., F.A.C.S.* 157

A Simple Oral Examination Light.....*Jack C. Cotton, Ph.D.* 163

Treatment of Stuttering.....*George A. Kopp* 165

The Treatment of Stuttering.....*Wendell Johnson* 170

Can a Child Be Taught To Talk?.....*Sara Stinchfield Hawk* 173

Society of Progressive Oral Advocates..... 180

Speech, Voice, and Hearing Bibliography,
Jeannette Dean-Throckmorton, M.D. 181

The JOURNAL OF SPEECH DISORDERS is published quarterly in March, June, September, and December of each year by the American Speech Correction Association; $1.00 a copy; $3.00 a year (Canada and foreign, $3.50). All communications and subscriptions should be sent to the editorial and business office: Speech Clinic, 324 Derby Hall, Ohio State University, Columbus, Ohio.

Entered as second-class matter at the Post-Office in Columbus. Ohio, under Act of Congress, of March 3, 1879.

PRACTICAL ASPECTS OF SPEECH CORRECTION*

MAX A. GOLDSTEIN, M.D., LL.D.
St. Louis, Mo.

The correction of defects in speech, handicapped and distorted by charlatans, commercial stammering institutes and inadequately qualified teachers, has now reached the dignity of a legitimate science, due largely to the research and earnest and untiring efforts of pioneers in various fields of medicine and pedagogy at home and abroad.

From the time of Demosthenes, the silver tongued orator of Greece who cured himself of stammering by juggling handfuls of pebbles in his mouth, to almost our own era when Dieffenbach, a German surgeon, in 1841, proposed an operation for resection of the tongue for the radical correction of stuttering, literature and history have jumbled facts and fancies associated with these human imperfections. The stammerer and the aphasic have stumbled through the ages beset by the accusations of witchcraft and mysticism, suffering as the scapegoats of ignorance and fanaticism and ostracised from social intercourse by the mental impotence and helplessness of their fellows to provide ways and means for overcoming such defects.

As knowledge was unfolded in anatomy and physiology of the speech mechanism, in neurology and the significance of cerebral localizations, in psychology and the relation of mental to physical stimuli, the fundamental principles were created for the establishment of a new science—the study of speech pathology and the classification and correction of defects in speech.

It is not my purpose to trace the evolution of this comparatively young science through its periods of struggle and misunderstanding. Let us rather view it today as a healthy young structure, built by sincere workers in medical and pedagogical sciences drawing on many fields including anatomy, physiology, neurology, psychology, the physics of sound, phonetics, the art of expression and the philosophy of language, for better understanding and development.

The earliest record in scientific literature of reference to and description of one of the most common forms of defects in speech (stammering), is found in the classical monograph of Hieronymus Mercurialis: De Mobis Puerorum Tractatus" a treatise on Diseases of Children, containing the first monograph on speech defects, published in 1583.

*Read at the meeting of the American Speech Correction Association, Cleveland, December 27, 1938.

In several chapters of this book he discusses the question of stammering as then conceived and refers, even though somewhat vaguely, to its scientific aspects. It is interesting to note that even in this early period, psychology and mental philosophy were receiving a share of consideration in an attempt to analyze this problem. The first impetus to such analysis was created by members of the medical profession.

More than five per cent of all school children in the U. S. A. have some form of defect in speech including stammering, stuttering, lisping, lolling, oral inactivity and various aphasias.

Within the past decade surveys have been initiated by public school systems of our larger metropolitan communities and ways and means established to organize special classes, qualify special teachers and provide special funds to rehabilitate these handicapped children.

As yet there has been comparatively little concerted action taken or special educational measures adopted in state or nation for the adequate care and training of this large group of children.

An important forward step has been taken by a number of our universities in the establishment of special departments and the appointment of specially qualified teachers to develop the problem of speech correction. In some instances this work has been assigned to the department of psychology; in others, to the department of English; in a few instances, an independent department of speech has been created in order that these activities may not be confused with other academic classifications.

Speech Clinics have been established in conjunction with universities in New York, Chicago, St. Louis, San Francisco, Iowa City, Madison, Columbus, and other centers where the classification, diagnosis and remedial treatment and pedagogy of various types of speech defects receive serious study and consideration in the classroom, clinic and laboratory.

The speech correctionist and the speech pathologist are not synonymous terms in character or in fact. The teacher of oral English, rhetoric, dramatics and public speaking is the speech correctionist, a numerical and formidable entity and a necessary factor in every school and in every grade, with opportunities for training and qualification in nearly every college and university. The speech pathologist is still a recent acquisition in the formidable fields of pedagogy and science, a minority group, limited in training and experience, with a wide and comparatively unexplored field of work and research in the offing.

Opportunities for the speech pathologist in his important and highly specialized field, are increasing daily; universities and colleges are beginning to recognize the value and significance of his endeavors; facilities in curriculums and clinics are being developed for his qualification and the dawn of a new era for the speech pathologist is nigh.

In an address by our guest speaker at the joint session of this association and the National Association of Teachers of Speech, the problem of plastic surgery in the correction of congenital defects of

the palate as an initial step in the rehabilitation of this large domain of speech defects and the work of the speech pathologist to make the subsequent speech corrections, will be given due prominence.

For many decades we have been empirically taught that changes in position of the complicated musculature of the tongue in its relationship to the oral cavity is an indispensable factor in the production of the consonant elements of oral speech.

To illustrate my contention that much of our knowledge of speech, as far as it concerns the functions of the tongue, is based on such empiricisms rather than on actual scientific evaluation, I offer accurate phonographic records of several cases where the tongue has been totally extirpated and where the subsequent speech of the subjects is still found to be distinctly understandable and of practical service.

The science of acoustics, the improved facilities of phonographic recording of speech and its finer analysis, the motion picture, radio and telephone engineering and similar agencies so useful to the speech pathologist in the furtherance of his observations, are making rapid strides and throwing new light on many of these confusing issues of anatomical and physiological data concerned with speech correction.

I have selected for presentation, a short movie film of a close-up illustrating unusual motility and gymnastics of various muscles of the tongue.

Literature on the physiology of the tongue is scant indeed, and there is but one substantial monograph on this subject presented within the last half-century.

A few years ago I demonstrated before the Society of Progressive Oral Advocates a male patient whose larynx had been extirpated and who showed his facility in the use of the artificial larynx not only in the fluency and ease with which speech was produced but also where voice inflection covering at least three or four intervals of pitch had also been accomplished.

This man, like the two patients who developed speech minus a tongue, was self taught.

To those familiar with the mechanism of the artificial larynx with its vibratory element composed of a tempered bronze reed, it is apparent that it is a difficult accomplishment to acquire pitch variation with such a rigid mechanism.

A better understanding of many of the less complicated pathologies of speech may be realized when we consider that such pathologies have their origin in defective hearing. The examination of many school children for hearing defects forcibly illustrates this observation and attention to and improvement in these hearing defects will often remedy a speech handicap without further psycho-analysis, environmental factors or long continued pedagogy.

Congenital aphasia has been admitted in the literature as a possible entity, but was not practically diagnosed until fourteen years ago. Before that time such cases, when seen at a very early age, were always accepted and taught as congenitally deaf children. Today it is possible to train a young aphasic of this type (especially

those with normal hearing) so successfully that he may carry on with a complete development of speech.

Traumatic aphasia is another unusual form to tax the speech pathologist's resources and abilities to the utmost.

I recall the case of an artillery officer in the World War who fell from his caisson and was kicked in the left side of the skull by a mule. A fracture of the left inferior frontal and parietal areas of the skull ensued and an amount of cortical brain substance from this area escaped. After plastic surgery of the skull had been completed he was returned home and referred for speech training. His central speech production or Broca area in the cerebral cortex had been so seriously encroached upon that his speech mechanism no longer functioned.

A detailed course of voice building and articulation similar to that used in the case of a congenitally deaf child, was planned for him. Within nine months this daily intensive training or re-education resulted in a complete restoration of his speech faculties.

Another case worthy of special comment in a World War veteran is that of speech restitution following destruction of the vocal cords.

An A. E. F. soldier was struck in the left side of the neck by fragments of shrapnel; part of the left mandible and tissues of the neck were destroyed, and the left thyroid cartilage of the larynx seriously mutilated. After the jaw had been repaired and the soft tissues healed, it was found that he had completely lost the use of his voice. Laryngoscopic examination revealed that the left vocal cord, together with the floor of the left ventricle had been completely obliterated and that the cicatricial tissue and healing process had involved the right cord as well—only a fragment of it could be seen imbedded in the thyro-arytenoid muscle. There were no vocal ligaments to function and there was no voice production. The area of the ventricular bands seemed undisturbed by this cicatricial process.

This ex-soldier received daily intensive training by breathing and speech exercises and was coached to produce sustained voice elements as though the true vocal cords were still present. Within one year he was demonstrated before a medical and pedagogical group and was able to speak and even to sustain tone in singing "The Holy City" and "The Long, Long Trail." His voice was husky, but of serviceable quality; his articulation was perfect; his speech easily understood and in volume could carry to the rear of the auditorium.

What voice compensation was accomplished in this case? Repeated laryngoscopic examinations showed an approximation of the vocal cords on phonation, and it is to be surmised that the function normally allotted to the vocal ligament was carried out by an approximation to the median line of the free triangular margins of the ventricular bands and their adjacent muscles.

These cases, selected from my personal experiences, emphasize and corroborate the value of my observation that much of the normal mechanism in speech production is still imperfectly or incompletely understood and that physiological compensation is a working asset offering opportunities for much research and closer study.

PALATOGRAPHY AND SPEECH IMPROVEMENT*

ELBERT R. MOSES, JR., Ph.D.,
Ohio State University,
Columbus, Ohio

INTRODUCTION

The object of this study is to determine whether there are fixed and definite tongue-palate contacts for various sounds, which may be utilized in speech improvement. Previous experimenters—Rousselot, Scripture, Panconcelli-Calzia, Navarro-Tomás, and others—would, through their publications, lead one to believe that certain sounds have a definite pattern of tongue-palate contact, regardless of individual differences, or other factors, among subjects. L'Abbé Rousselot thought that brogue might be corrected by differences in tongue position. James L. Barker, in his article, "Correcting the Mechanism Causing Foreign Brogue," mentions the above fact, but maintains that during a period of five years Rousselot did not have a single success in eliminating English brogue in the pronunciation of French.[1] If the claims of Rousselot and other earlier phoneticians were true, palatography would be an invaluable aid in speech improvement instruction.

James L. Barker thinks that the important factor in pronunciation is movement.[2] He is practically alone among phoneticians, however, for the rest seem to consider position as the major if not the sole factor. Navarro-Tomás is probably representative of these. In his *Manual de Pronunciacion, Española*, he speaks, it is true, of American pronunciation of the Spanish final *z*'*s* and *s*'*s* as having excessive force and duration, but makes no comment on how one goes into position or out of position. By and large, his general view is probably best represented by his discussion of the *t*. He mentions that the point of articulation of the French *t* is lower than that of the Spanish *t*, and

*The palatographic material for this study is derived from a thesis, *Palatography—A Critical Study and Analysis of Contacts*, which was completed under the direction of Clarence L. Meader, John H. Muyskens, Leon L. Strong, Hide Shohara, Louis M. Eich, in partial fulfillment of the requirements for a Ph.D. degree in the Horace H. Rackham School of Graduate Studies, of the University of Michigan, June, 1936.

[1] *Journal of Speech Disorders*. September, 1937. p. 165.

[2] "Thus the difficulty (overcoming brogue) is one of habit, not of taking certain positions or maintaining them, but of movement in relation to breath-control; it is a difficulty that concerns, not static phonetics, but dynamic phonetics." "Correcting Foreign Brogue," *Journal of Speech Disorders*, March, 1936. p. 11.

that the German and English *t's* are articulated farther up against the gums and alveolar ridge, which, added to the aspiration which ordinarily accompanies the voiced occlusives, gives to the *t* a timbre which is quite different from that manifest in Spanish,[3] and he accompanies his observations by a palatogram of one Spanish *t* of one subject, leaving the impression that this would be characteristic of all. He makes no reference, however, to movement; his work is purely of a static nature.[4] He states that in order to pronounce the Spanish *t* as differentiated (moving away) from the English *t*, it is necessary on the one hand to advance the point of the tongue more than one centimeter toward the teeth, and on the other to cause the explosion of the consonant to be free and clear (limpia y sonora) without any friction or aspiration whatsoever.[5] He has not dealt with the length of the *t*, as it is moved into or out of position; and he apparently believes that in palatography position is the prime factor.

Sir Richard Paget believes that differences in consonants are of a harmonic nature.[6] Henry Sweet mentions the "on glide" and

---

[3]"A la especial posición adoptada conjuntamente por dichos órganos en el momento de producir un sonido, se le llama articulación; al movimiento . . . también; pero en el presente libro esta palabra va siempre empleada en la primera de ambas aceptiones." p. 16–17, 4th edition. Madrid, 1932.

[4]In one instance (*Manual de Pronunciacion Española*, 4th edition, 1932, p. 96) in his discussion of *t* and *d*, Navarro-Tomás shows in median sagittal section the tongue tip touching the alveolar ridge and gums; while the corresponding palatogram shows the tongue touching completely around the outer edge of the hard palate. This is characteristic of his treatment without consideration of the movement to and from position of articulation.

See Plate II (*t* and *d*) of this study for a comparison of tongue positions for the articulation of the American *t* and *d*.

[5]In speaking of foreign brogue in the pronunciation of a *t*, his sole statement is: "La *t* francesa tiene su punto de articulación un poco más arriba que la española. La punta de la lengua en la *t* francesa no toca los bordes de los dientes. La *t* alemana y la inglesa se articulan aún más arriba que la francesa, contra las encías y los alvéolos, lo cual, unido a la aspiración que en estos idiomas acompaña ordinariamente a las oclusivas sordas, da al sonido de la *t* un timbre muy distinto del que presenta en español. La *t* inglesa con su articulación alveolar o postalveolar es la que más se aparta de la nuestra. Especialmente en aquellos casos en que va agrupada con una *r* siguiente, en palabras como *cuatro*, nuestro, tropa, etc., la *t* que pronuncian los ingleses y los angloamericanos llega a presentar un sonido completamente extraño a la pronunciación española normal.

"Entre los defectos de pronunciación que los extranjeros necesitan evitar para hablar español correctamente, el que se refiere al punto y al modo de articulación de la *t* es uno de los más importantes. Para pronunciar la *t* española, partiendo de la *t* inglesa, es necesario, de una parte, hacer avanzar la punta de la lengua más de un centímetro hacia los dientes, y de otra, hacer que la explosión de la consonante resulte limpia y sonora, sin fricación ni aspiración perceptibles. *Manual de Pronunciacion Española*, 4th edition. Madrid, 1932. pp. 97–98.

[6]"Consonants are due to resonance like the vowels; but inasmuch as they are produced by movements of the vocal organs (like diphthongs) their resonances are characterized not only by pitch, but also by their change and rate of change in pitch. The consonants are all made by a complete or partial closure of the front or back orifice of the front resonator of the series which produces them. In every case there are more than two resonators in action." *Human Speech*, pp. 124–125.

"off glide" of voiced consonants between vowels, or the movement into position and the movement out of position[7] but he, too, is most concerned with the position itself.

Dr. G. Oscar Russell believes, as does Barker, that other factors besides tongue position may be involved. At least in one case he refers particularly to type of tongue movement.[8] More recently, he, in conversation with the author, has summarized the various factors he would consider as being vital, as follows:

(1) length of time the consonant is held,
(2) the way one moves into position,
(3) the way one moves out of position,
(4) the amount of breath pressure behind the consonant,
(5) position of the vocal organs,
(6) types; whether voiced or unvoiced, occlusive, fricative, affricative, nasal, or liquid.

All of the preceding information is of interest to the author and reader because our present study seems to show that there is no fixed tongue position for any given sound made even by a single individual at various times, much less by different people in the same language group. It tends also to substantiate Barker's statement that foreign brogue depends for its correction upon "changes in habits of movement and breath-control indicated and none of them may be corrected by a system of phonetics based on a study of static positions alone."[9]

This study involves two palatography projects. The first project was carried out in a class in dynamic phonetics at the University of Michigan in 1933 under the direction of Dr. John H. Muyskens, now Director of the Horace H. Rackham Clinic. This project later became the first of three projects of a doctoral thesis.[10] The second project involves a group of palatograms made by means of a new technique for the thesis study.[11] Both projects tend to prove that there is not a static position of the tongue for the pronunciation of certain sounds.

---

[7]"Such a word as *cat* consists not only of the vowel and the two consonants of which it is made up, but also of glides or positions between these sounds. The glide from the initial consonant to the vowel consists of all the intermediate positions through which the tongue passes on its way from the *k* position to that of the æ. The number of these positions is infinite; but they are all implied by the mere juxtaposition of the symbols of the fixed sounds, it being assumed that in all transitions from one position to another the shortest way is taken." *Sounds of English*, p. 54.

[8]"The Pronunciation of the Spanish *R*." *Modern Language Journal*, III: 174–184, 1919.

[9]"Correcting the Mechanism Causing Most Foreign Brogue," *Journal of Speech Disorders*, March, 1936. p. 12.

[10]See author's thesis, "Palatography—A Critical Study and Analysis of Contacts," unpublished, in the University of Michigan Library, 1936.

[11]This project was designated as two projects in the thesis (Projects II and III).

APPARATUS AND PROCEDURE

Palatograms for the first project were prepared according to the method then current in the University of Michigan Speech Laboratory. With Kerr's Impression Compound an impression of the hard palate was made. This impression was boxed with boxing wax; and into the bowl thus formed was poured a mixture of Castone (plaster preparation). Into the palate of the cast was swedged a square of ash metal No. 7 until the metal assumed the contour of the palate; then the metal was trimmed off around the teeth of the cast. Next the artificial or pseudo-palate was painted with jet-black enamel. By placing the pseudo-palate hollow-side-downward upon a piece of cardboard, an outline was made of the palate area. This outline of the pseudo-palate, cut from the cardboard, formed the pattern for prints of the pseudo-palate to be made on oil-skin tracing paper. Numerous sheets containing this outline, or pattern, of the palate were prepared. On the enameled surface of the pseudo-palate unscented talcum powder was dusted, the palate was inserted into the mouth, the sound articulated, and the pseudo-palate hastily but carefully removed. This tongue-contact picture was quickly drawn upon the palate-print on the prepared sheets of paper. That part of the palate on which the powder remained was blackened with pencil in the picture. After the tongue contact had thus been transferred to the paper, the sound symbols were written on the reverse side of the paper. It was from this side that the sheets later were blue-printed. In this manner the resulting blue-print pictures were identical with the tongue-contact pictures appearing on the pseudo-palate. This procedure was followed by each individual participating in the project.

In this present form of the study, I have included Russell's additional technique of an exact centimeter measurement plan for showing on a flat surface the perpendicular arch of the palate.[12] Accordingly, a base-plate model of the hard palate was made.[13] Beginning at the median line of the palate, I marked off the entire palate surface by a series of pin-holes. These dots were located along parallel and right angle lines at intervals of one centimeter, thus blocking the whole off into units of one square centimeter. The palate then was turned over, a paper was placed on top, and the light shining through the pin-holes allowed me to trace the dots on paper. The result, with the dots in this arrangement, gives one an idea of the arch and the irregularities of the palate, because wherever the palate surface slopes the dots will be less than one centimeter apart, how much less being dependent upon the steepness of the slope. After preparing on tracing paper a series of master models for the subjects used in this experiment, I traced onto the master models from the original blue-print palatograms the tongue-palate contacts. These final palatograms were reduced to half-size for printing purposes. Refer to Plates I, II, III.

[12]This method is mentioned by Dr. G. Oscar Russell in *The Vowel*, p. 335ff.

[13]I had kept on file the original impressions from this experiment.

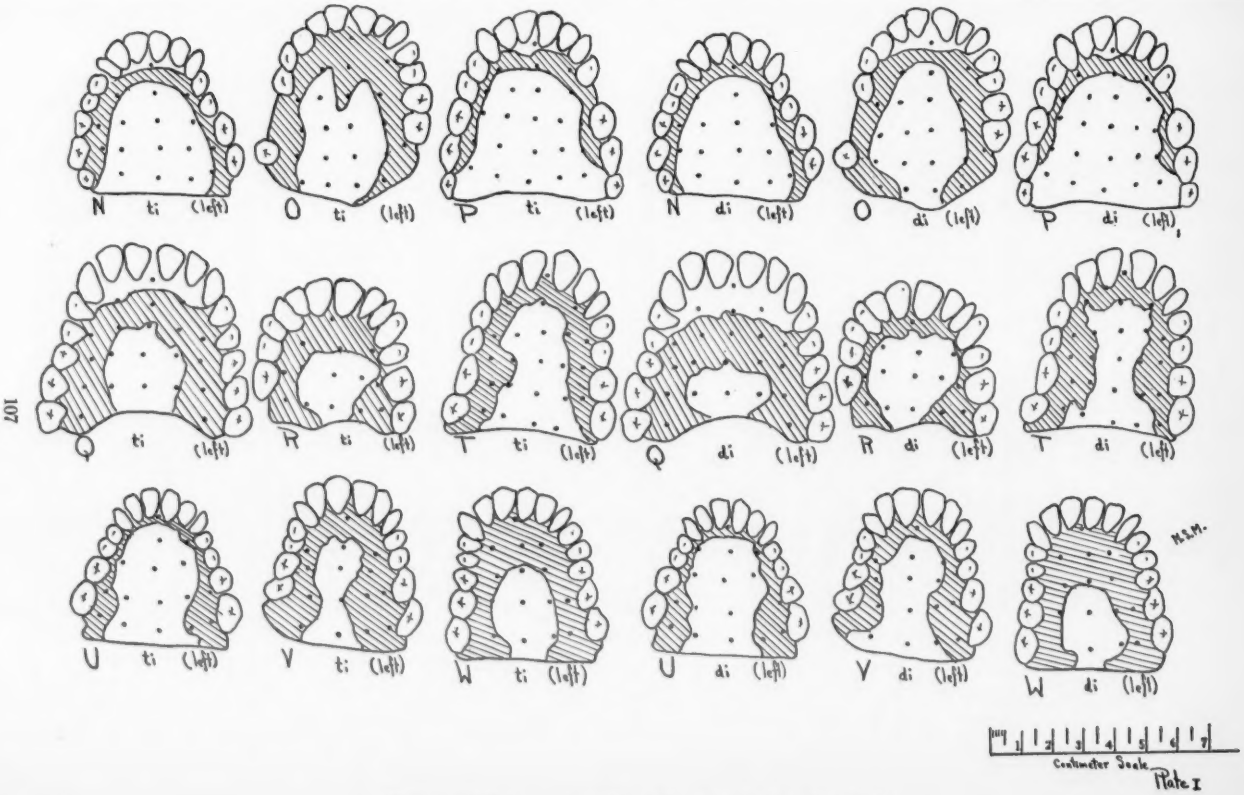


Plate I

(See page 108 for explanation of Symbols used in this plate)

In the second project a new technique was employed. I prepared numerous casts of each student's palate.[14] To show the depth of the tongue contact on the hard palate, these casts were cut in half from the division between the medial incisors to the soft palate. I used the pseudo-palate described above. Each sound or sound combination was made three times by each subject,[15] and these three contacts were outlined, each in a different color, on the split casts. (An outline was used because, after considerable experimentation, it was found impractical to super-impose one color upon another in an effort to designate the entire area of each of the three repetitions.) In transferring the contact pattern from the pseudo-palate to the positive cast, the pseudo-palate was placed in the palate of the cast; certain directional marks were made on the cast; and, with the aforesaid marks as guides, and with calipers to measure the width of contact, an outline of the contact as shown on the pseudo-palate was drawn upon the positive cast.

When the subjects had completed the sound combinations, the marked casts were photographed so that a permanent and usable record of the contacts might be made.

SUBJECTS AND SOUND COMBINATIONS

Nine subjects, to be designated as subjects N, O, P, Q, R, T, U, V, W, participated in the first project, each completing a set of palatograms for the following sound combinations:

(The symbol "a" is used for the phonetic symbol "ae" in all examples throughout this study.) ad, at, da, ta, ɛd, ɛt, dɛ, tɛ, ɪd, ɪt, dɪ, tɪ, ag, ak, ga, ka, ɛg, ɛk, gɛ, kɛ, ɪg, ɪk, gɪ, kɪ, az, as, za, sa, ɛz, ɛs, zɛ, sɛ, ɪz, ɪs, zɪ, sɪ, aʒ, aʃ, ʒa, ʃa, ɛʒ, ɛʃ, ʒɛ, ʃɛ, ɪʒ, ɪʃ, ʒɪ, ʃɪ, la, al, lɛ, ɛl, lɪ, ɪl, ra, ar, rɛ, ɛr, rɪ, ɪr, ab, ap, ba, pa, ɛb, ɛp, bɛ, pɛ, ib, ip, bi, pi, av, af, va, fa, ɛv, ɛf, vɛ, fɛ, iv, if, vi, fi, aδ, aθ, δa, θa, ɛδ, ɛθ, δɛ, θɛ, iδ, iθ, δi, θi, ab, ba, ap, pa, ɛb, bɛ.

A history of each subject was recorded as follows:

Subject N.
Age—23 years. Nationality of parents—Filipino.
Languages spoken other than English—Philippine dialects.
Languages studied in school—English, Spanish.
Undergraduate scholastic average—C.

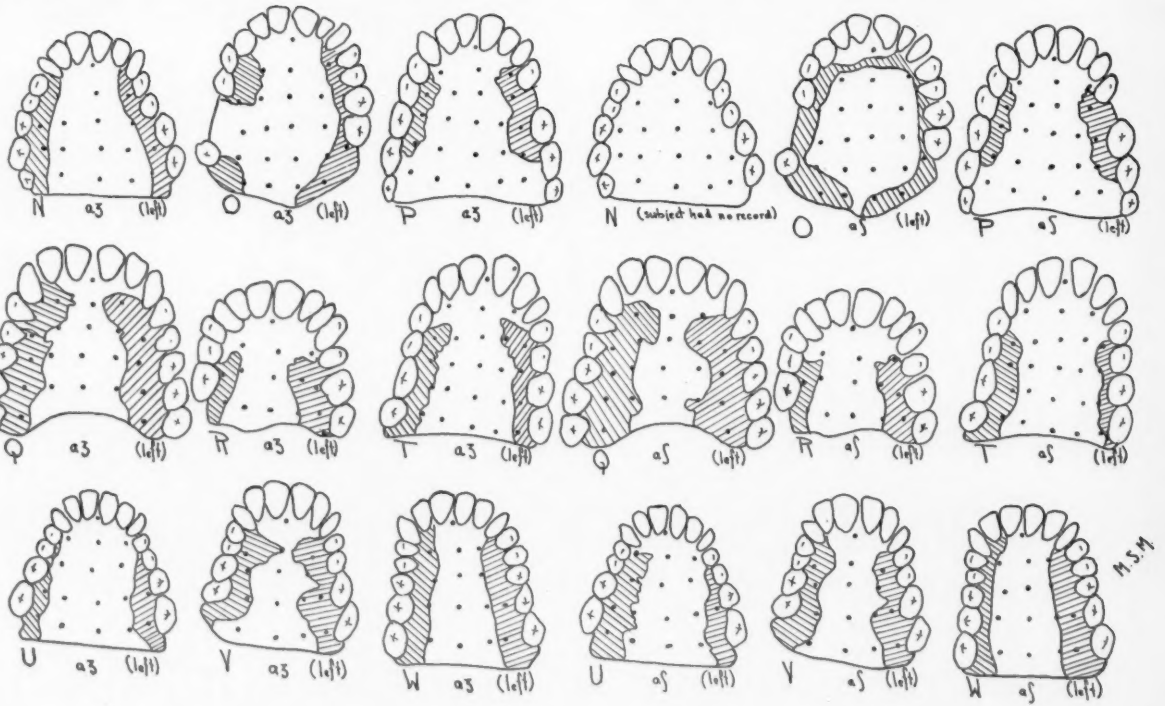
Subject O.
Age—38 years. Nationality of parents—English.
Languages spoken other than English—none.
Languages studied in school—Greek, Latin, Hebrew, French.
Undergraduate scholastic average—B.
This subject was an instructor in speech in a college.

Subject P.
Age—26 years. Nationality of parents—On father's side, English and Irish; on mother's side, German and Swedish.
Languages spoken other than English—none.

---

[14] I discovered that by the use of the dental preparations, *Dentocol* and *Reprolastic*, and the sculptor's method of glue-mold casting, I could produce any number of casts desired from the one original negative impression.

[15] See the author's thesis, appendices III, IV, V.



(See page 108 for explanation of Symbols used in this plate)

Languages studied in school—Latin.
Undergraduate scholastic average—C.
Remarks: Post-operative cleft-palate speech.

Subject Q.

Age—25 years. Nationality of parents—English.
Languages spoken other than English—none.
Languages studied in school—French, Spanish, Russian, Latin.
Undergraduate scholastic average—B+.

Subject R.

Age—27 years. Nationality of parents—American.
Languages spoken other than English—none.
Languages studied in school—French, Latin.
Undergraduate scholastic average—B+.
Remarks: Had taught diction and dramatics.

Subject T.

Age—21 years. Nationality of parents—Father, German; mother, English.
Languages spoken other than English—none.
Languages studied in school—French, Latin, German.
Undergraduate scholastic average—C.

Subject U.

Age 17 years. Nationality of parents—German, Russian.
Languages spoken other than English—none.
Languages studied in school—Latin, Greek, French.
Undergraduate scholastic average—B—.

Subject V.

Age—20 years. Nationality of parents—English, Irish.
Languages spoken other than English—none.
Languages studied in school—French, Latin.
Undergraduate scholastic average—A—.
Remarks: This subject had 36 hours of speech study.

Subject W.

Age—20 years. Nationality of parents—American.
Languages spoken other than English—French, German.
Languages studied in school—French, German, Latin, Italian.
Undergraduate scholastic average—C.

Three subjects, to be designated as Subjects E, B, M, participated in the second project, each completing a set of palatograms for the following sound combinations: ad, at, da, ta, ɛd, ɛt, dɛ, tɛ, ɪd,, ɪt, dɪ, tɪ, ag, ak, ga, ka, ɛg, ɛk, gɛ, kɛ, ɪg, ɪk, gɪ, kɪ, ða, θa, da, ta, ga, ka, za, sa, að, aθ, ad, at, ag, ak, az, as, ði, θi, di, ti, gi, ki, zi, si, az, as, za, sa, ɛz, ɛs, zɛ, sɛ, ɪz, ɪs, zɪ, sɪ, aʒ, aʃ, ʒa, ʃa, ɛʒ, ɛʃ, ʒɛ, ʃɛ, ɪz, ɪʃ, ʒɪ, ʃɪ, la, al, lɛ, ɛl, lɪ, ɪl, ra, ar, rɛ, ɛr, rɪ, ɪr, ɪð, iθ, id, it, ig, ik, iz, is.

The histories of these three subjects are as follows:

Subject E.

Age—27 years.
Nationality of parents—American.
Languages spoken other than English—none.
Languages studied in school—Latin, French, German.
Undergraduate scholastic average—B.
Remarks: Had taught diction, dramatics, voice; was a graduate student in speech.

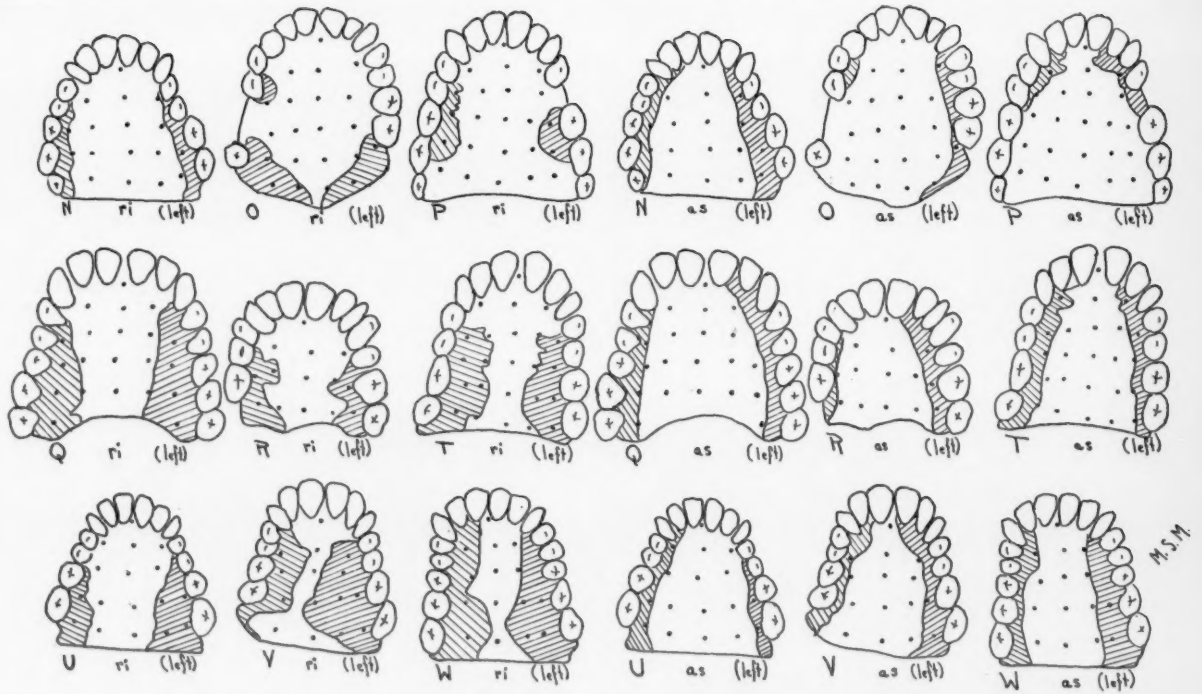


Plate III

(See page 108 for explanation of Symbols used in this plate)

Subject B.
Age—27 years.
Nationality of parents—American.
Languages spoken other than English—none.
Languages studied in school—none.
Undergraduate scholastic average—Did not have a college education.

Subject M.
Age 28 years.
Nationality of parents—American.
Languages spoken other than English—none.
Languages studied in school—French, Spanish, Latin.
Undergraduate scholastic average—A—.
Remarks: The subject held a Master's degree in history; had taken some work toward a doctorate in speech; had done considerable technical work in speech experiments.

RESULTS AND DISCUSSION

When the nine students had completed the first project, I had in my possession 864 palatograms covering 102 sound combinations. Of this number, some were found impractical for use in an experiment such as this one. The sound combinations ab, ba, ap, were found not to give any palatal impression, for there is no contact between the tongue and the palate in the articulation of these sounds. The combinations eb, pa, be, showed contacts in some instances: pa in 2 cases out of 9; be in 6 cases out of 9; eb in 6 cases out of 9. Of this group showing contacts where others failed to show contacts, only one subject had a speech abnormality. It is plausible, therefore, to state that certain vowel-consonant combinations produce contacts in some instances and not in others, depending upon the individual articulating the sound.

The second project involved 276 photographs of split palate casts, covering 92 sound combinations.

Selection of the palatograms appearing on Plates I, II, III, was made at random. Plates I, II, III, show the following contacts for aʃ, aʒ, id, it, as, it, the same sound being made by each of the nine subjects.

Plate IV shows subjects E, B, and M each making the same vowel-consonant combination three different times in successive articulations. After each articulation the pseudo-palate was removed so that the pattern might be drawn upon the cast. The plate shows the following vowel-consonant combinations: ɛk, kɛ, ga, ka, ar, ra.

Even a cursory examination shows that no one of these subjects makes tongue contacts for the vowel-consonant combinations which are the same, or even generally the same, either as compared with the others or from one of his trials to the other. They all vary considerably. The same type of variation occurred in the majority of the contacts made by these same subjects.[15] In those vowel-consonant combinations which were identical in the two projects, each subject of Project II made contacts quite different from those made by any one of the other subjects, N, O, P, Q, R, T, U, V, W, all of whom also differed from each other. It may be concluded from

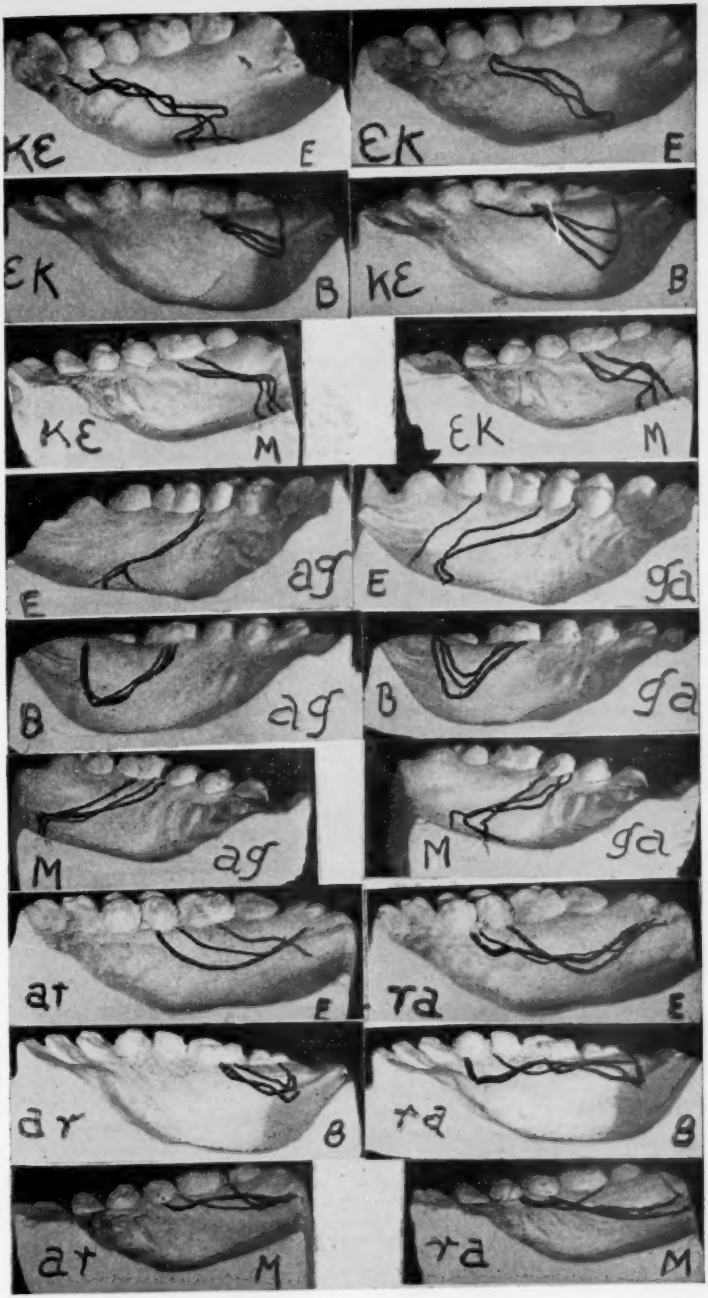

KE
E
EK
E
EK
B
KE
B
KE
M
EK
M
E
ag
E
ga
B
ag
B
ga
M
ag
M
ga
aT
E
Ta
E
aT
B
Ta
B
aT
M
Ta
M

PLATE IV

1140 tongue-palate contacts of 13 subjects[16] in 102 sound combinations, that:

> no two persons make the same tongue-palate contact for the same sound or sound combination, and that no person makes the same tongue-palate contact for the same sound in the same way, even on the same day.

CONCLUSIONS

In view of the above results, it may be concluded that

1. In the correction of foreign brogue or speech defects, it is not justifiable to say that any so-called correct tongue position for any consonant can be indicated exactly.
2. There is a possibility not only that different persons speaking the same language do not make the same tongue-palate contact for the same sound or sound combinations; but that the same persons do not make the same contact for the same sounds, even on the same day.[17]

These conclusions infer that in the correction of foreign brogue and speech defects, palatography may be used, but that it cannot be used so extensively as it has been by phoneticians in the past. Because of other factors, already mentioned, it cannot be said that in the articulation of a given sound or sound combination the tongue should strike the palate at a given place in a given contact pattern. Needless to say, factors other than position must therefore be involved; and these, too, must therefore be given more attention while position is given less than has been customary in the past.

BIBLIOGRAPHY

Navarro-Tomas, T. *Manual de Pronunciacion Española.* 4th edition. Madrid, 1932.

Paget, Sir Richard. *Human Speech.* Harcourt Brace and Company. Boston, 1930.

Panconcelli-Calzia. *Die Experimentelle Phonetik in Ihrer Anwendung Auf Die Sprach-Wissenschraft.* Verlag Van Walter De Gruyere & Company. Berlin, 1924.

Rousselot, L'Abbé P. J. *Principes de Phonétique Expérimentale.* Nouveau ed. Paris. Libraire Universitaire. 1910.

Russell, G. Oscar. *The Vowel.* Ohio State University Press, Columbus, Ohio. 1928.

Scripture, Edward Wheeler. *Experimental Phonetics.* Charles Scribner's and Sons. New York, 1902.

Sweet, Henry. *Primer of Phonetics.* 3rd ed. Oxford University Press, 1906.

Sweet, Henry. *Sounds of English.* Oxford University Press, 1910.

Barker, James L. "Correcting the Mechanism Causing Foreign Brogue." *Journal of Speech Disorders,* September, 1937.

Barker, James L. "Correcting Foreign Brogue." *Journal of Speech Disorders,* March, 1936.

Russell, G. Oscar. "The Pronunciation of the Spanish *R.*" *Modern Language Journal,* III: 174–184. 1919.

---

[16]An additional subject was used in the thesis (Subject S, cuts found in Appendix V), but has not been included in this particular study.

[17]Statistical analysis of contacts is not a part of this study; the author, however, has made such a statistical study for the above-mentioned sounds in his thesis.

THE HISTORICAL BACKGROUND OF THE MODERN SPEECH CLINIC

G. M. KLINGBEIL

(From the Speech Clinic Seminar of Dr. G. Oscar Russell, Summer Session, University of Wisconsin)

In preparing this paper it has seemed advisable to divide the topic into three divisions, the first dealing with stuttering, the second with aphasia, and the third with other speech problems, including that of the deaf. The arrangement is, as far as possible, chronological, and since theories of etiology are necessarily closely connected with remedial practice, no attempt to separate them has been made. A bibliography is appended at the end of each section, but the title of the original writings of each respective practioner is given when reference is made to his theories.

STUTTERING AND STAMMERING

Herodotus (*c* **484-424 B. C.**) the Greek historian, records the treatment of Battos, son of Polymnestos, by a Pythian priestess, who recommended emigration south, to Libya.

Hippocrates (*c* **450-357 B. C.**) the Greek physician, the "Father of Medicine," remarked that there existed among stutterers a chronic diarrhoea—not of words—and described its cure by means of varices, which resulted in suppurating ulcers. *Praecept. 6; Aphor. 6, 32; Epidem. 2, 5, 6; De Judicat. 6.*

Aristotle (*c* **384-322 B. C.**) came from a Greek family in which the practice of medicine was hereditary. He trained himself as an orator and described a number of speech defects, and attributed the whole blame in stuttering to the tongue.

Satyrus the Greek actor, is said, by Plutarch, to have been responsible for the cure of *Demosthenes* (*c* **383-322 B. C.**) who not only had a harsh and tuneless voice, but stuttered and had difficulty with the pronunciation of "r." Satyrus prescribed voice exercises for him, in which the use of a mirror and pebbles in his mouth were part of the treatment, which called also for declaiming while going uphill. *Chervin* (*Ainé*), the French specialist of the nineteenth century, claims that Satyrus was the first to combat stuttering by lessons of diction. M. Chervin—"*Du Bégaiement considéré comme vice de prononciation.*" Paris **1867.**

Cicero, Valerius Maximus, and **Catullus,** writing in the **1st cent. A. D.** all mention various defects of speech.

Celsus, Aurelius Cornelius (B. C. 42-A. D. 37) compiled encyclopaedic treatises on medicine, and presumably rendered medical treatment gratis. Though ignored by the Roman practitioners of his time, his book, *De Re Medica,* was printed and came into its own in **1478.** The 6th volume deals with diseases of the eye, ear, nose, mouth and throat. He advocated the use of gargles for faulty articulation rising from disease of the tongue, or other cause; massage of the tongue and throat, the eating of pungent substances, and performing physical and respiratory exercises, and the economising of breath during speech.

Galen (131-201 A. D.) practised cauterization.

AEtius of Amida, who lived in the **6th cent.,** and was royal physician to Justinian at Byzantium, left an extensive compilation usually called the *Tetrabiblion.* the first eight books of which were published in Venice in **1534.** His work is said to contain the best account of diseases of the eye, ear, nose, throat and teeth in the literature of antiquity. He blamed the tongue as the cause of stuttering, and recommended division of the frenum. *Ætii graeci contractae ex veteribus medicinae Tetrabiblos. Basiliae* **1542** chap. xxxvi.

AEgineta, Paulus, was a Greek physician who lived in the latter half of the **7th cent.** and traveled much. His medical works were highly prized, though they were mainly compilations from earlier writers. He also laid the blame on the tongue.

Avicenna, Abu Ali Al Husain (980-*c.* 1037) was a philosopher and court physician of Arabia. He travelled much and compiled the "*Kanun fi'l tebb*" a system of medicine based on an Arabic translation of Greek medical works, which became the text book of both East and West for many years. He laid the blame for stuttering largely on the tongue, but blamed also lesions of the brain or nerves, and sometimes spasm of the epiglottis, which latter he advised should be treated by taking a deep inspiration before speaking. *Avucebbae orubucous etc. a Joanne Paulo Mongio et Joanne Costaeo recognita.* Venetiis, **1564.**

De Chauliac, Guy (1300-*c.* 1380) was a French surgeon and physician to three popes at Avignon. His *Chirurgia Magna* **1363** was for three centuries an indispensable manual. He ascribed stuttering to convulsions, ulcers, or other affections of the tongue; to paralysis, or to moisture of the nerves or muscles. His treatment consisted of embrocations to dessicate the brain, cauteries for the vertebrae, blisters, frictions and gargles for the tongue.

Mercurialis, H. (1530-1606) professor at Padua, Bologna, and Pisa, attributed the cause of stuttering to either abnormal dryness or moisture of either the brain or the tongue. He followed the treatment of *De Chauliac* with the addition of systematic exercise of the voice and body.
De Puerorum Morbis. Francofurti, **1584.**

Hildanus (1608) operated on his brother for a defect of speech by cutting the frenum of the tongue.

Bacon, Francis Lord (1560-1626) attributed the cause of stuttering to coldness and moisture of the tongue, or, occasionally to its dryness.
Sylva Sylvarum, **1627.** cent iv sec. 386.

Menjot, Antoine (*c.* 1615-1696) who practiced medicine at Montpelier, and was later physician to the king at Paris, wrote a comprehensive treatise on stuttering and other speech defects, blaming a tongue abnormality for all of them.
Dissertatio Path. de Mutitate et Balbutie. Paris **1674.**

Amman, Johann Konrad (1667-1724) was a Swiss physician and one of the earliest writers on the instruction of the deaf and dumb. He practised in Holland, and also treated stutterers. He believed "hesitantia," as he called stuttering, to be due simply to vicious habit. He directed his treatment for it chiefly to the tongue.
Dissertatio de loquela. Amsterdam **1700.** *Surdus loquens* Amtserdam **1692.**

Kustner blamed the tongue, and directed his treatment almost wholly to it.
Dissertatio Inauguralis de Lingua etc. Altdorfii, **1716.**

Hahn, Johann Gottfried von (1694-17—) studied medicine at Leipzig, became a famous practitioner in Breslau, and later, court physician to King Frederick. He laid the blame for stuttering on the hyoid bone.
Commerc. Litt. **1736.**

Hartley was an English philosopher who gave up the church for medicine. His famous work *"Observations on Man"* deals in Part 1 with the constitution of the human mind, and in Part 2 with religion and morals. His treatment of the mind turns on the doctrine of vibrations, or a theory of nervous action analogous to the propogation of sound. He maintained that all conscious processes had physical correlations in the brain processes, and considered stuttering to be caused usually by nervous derangement though sometimes also to imitation, and often to palsy.

Haen, Anton (1704-1776) was an Austrian doctor whom Queen Marie Theresa called upon to bring about medical reforms in the country, and to be her private physician. He established the clinical method in Germany. In his opinion, pulmonary vomicae were the cause of stuttering.
Ratio medendi etc. Vienna **1705.**

Santorini, Giovanni Domenico (1681-1737) the Italian anatomist responsible for the discovery of the tubercles of cartilaginous knobs of the larynx, believed that an abnormal size of "two holes in the middle region of the palate" were the causative factor in stuttering.
Opuscula Medica etc. Venetiis **1706.**

Morgagni, Giovanni Battista (1682-1771) is regarded as being the founder of pathological anatomy. President of an independent medical school he later became Professor of Anatomy at Padua, and was the first to call attention to the false vocal bands, the Ventricle of Morgagni. He rejected *Santorini's* theory, and also that of *Delius* of a double velum, and inclined toward blaming the hyoid bone as the most likely offender.
De aurium et narium affectibus, aliquid additur de balbutie. Bonet's Sepulchretum. Sect 22 obs. xxi. Venetiis, **1755.** Lugduni Batav. **1761.**

Sauvages, F. B. S., considered the principle cause of stuttering to be debility, and recognized seven different kinds of Psellism or stuttering.
Nosologie Methodicus. Lyon **1772.**

Cullen, William (1710-1790) professor of medicine at Edinburgh and later at Glasgow, where he taught a system of nerve influence, also blamed debility for stuttering.
Synop. Nos. Med.

Mendlessohn, Moses (1729-1786) was born at Dessau, and is noted for his attempts to find good reasons for opinions currently held. He insisted that the chief reason for stuttering was psychical, and was due to a collision between many ideas flowing simultaneously from the brain. He recommended slow reading aloud, the succeeding words being kept covered, so as not to be seen until they were required to be enunciated.
Psychological observations on the case of Spalding. Magazin zur Erfahrungsseelenlehre. Berlin **1783.**

Crichton, Alex., gave a vivid description of stuttering and particularly emphasized the fact that there are but few sounds on which any one person stutters.
On Mental Derangement. London **1798.**

Darwin, Erasmus (1731-1802) English physician and naturalist, keen and philosophic observer of nature, believed that sensations or emotions such as awe or bashfulness caused interrupted association of the movements of the organs of speech. He advocated constant practice of the difficult sounds with as much softening of the initial consonants as possible.
Zoonomia. London **1800.**

Watson, J., ascribed stuttering to a disseverance of the associated action of the muscles, *i. e.*, want of co-ordinating power. According to him this "vicious habit" could be overcome by means of exercise of the organs of speech and strengthening of the will.
Instruction of the Deaf and Dumb. London **1809.**

Thelwall, John (1764-1834) was an English lawyer and according to *Potter*, a typical stutter-doctor. He considered the cause to lie either in the lips, tongue, pharynx or bronchi, and hence differentiated four different species, all of which he treated through rhythm.
Illustrations of English Rhythmus. London **1812.** *On Imperfect Development, etc.* London **1810.**

Savary was a French doctor who ascribed stuttering chiefly to malformation of the tongue, though fear, or hurried speech might occasionally be a contributory factor.
Dict. des Sciences Méd. art.—"Bégaiement" Paris **1812.**

Itard, J. M. G., was a French doctor who died in Paris in **1838.** He was a surgeon and later doctor for the Institute for Deaf and Dumb in Paris. He maintained stuttering to be due to muscular debility, and used a golden or ivory fork, placed in the cavity of the alveolar arch of the lower jaw, for the purpose of supporting the tongue. He reported but two cures, neither of which were permanent according to other accounts, which it is true may be biased and not based on actual observation.
Journal Universel des Sciences Méd. Paris **1817.**

Dupuytren, who lived about the same time as Itard (ca. **1817**), made his patients speak in a singing tone, marking certain intervals by a slight movement of the foot. *Colombat* (vide infra) is incorrectly regarded as being the originator of this technique.

Frank, Joseph, was a doctor who used an elaborate nomenclature for defects of speech, and enumerated many causes of stuttering, such as cerebral lesions, sexual excesses, etc. His treatment consisted wholly of exercises for the voice and speech, under the direction of a teacher of deaf-mutes.
De vitiis bocis et loquelae. Lipsiae, **1811.**

Rullier, P., was a doctor who ascribed a two-fold cause to stuttering, firstly a disproportion between the rate at which the brain produces thoughts, and that at which it transfers them to the different seats of innervation; and secondly an incapacity of the organs of speech to accomplish their tasks satisfactorily. The inability of the parts to cope with the demand made upon them when profusion of stimuli come by quick turns, is the cause of the spasmodic inflexibility of the stammerer's organs. The central organ is exclusively responsible for this spasm owing to its precipitance.
Dict. de Médicine Art. "Bégaiement." **1821.**

Voisin, Félix, a French doctor whose theory regarding etiology was similar to that of Rullier, being himself a stutterer, had a practical experience to guide him; yet his only contribution to the treatment was the trick of pressing the thumb against the chin while speaking.
Du Bégaiement, ses causes etc. Paris, **1821.**

Astrié, also located the cause in some modification of brain action. His treatment combined the study of articulation with recitation, and the use of Itard's fork.
Essai sur Bégaiement. Montpellier, **1824.**

Yates, early **1800,** was a New York doctor, and according to some, the inventor of the system known at that time as "the American Method." His treatment was carried out in an institute under the direction of "the *widow Leigh*" governess of Yates' daughter, this being done to avoid professional disrepute. Another version attributes the invention to *Mrs. Leigh* herself, whose husband had been a stutterer. Yates believed that stuttering was caused by spasm of the glottis, and treated it by advocating the raising of the tip of the tongue to the palate, and holding it there while speaking. The secret was bought by *Malebouche** who made it "scientific" and traded on it, selling it to the Prussian and Belgian governments. *Baussmann** was made "professor" of the system by the Prussian Ecclesiastical Minister of State, and *Dr. Zitterland* and *M. Charlier* were also prominent in its application

*Vide infra.

throughout Europe. It was made the subject of a report by *Magendie** to the French Academy in **1828,** and died a natural death shortly after having excited a phenomenal amount of attention. *Canon Kingsley** states that this method was secretly peddled by an English quack many years before.

Combe, George (1788-1858) born in Edinburgh, helped to found the Phrenological Society, and began to publish the Phrenological Journal in 1823. The cause of stuttering, in his opinion, lay in irregular nervous action, and he recommended the patient and persistent exercise of the vocal muscles with attention to diet and hygiene, together with cheerful society, kind treatment, and encouragement.
Phrenological Journal **1827**, p. 464.

Broster, John, was a Scotch "stutter-quack" who practiced in London and wrote a book on his method, but kept it as much in the dark as possible. His system is stated by *Dr. Julius* in the *Magazine of Foreign Medical Literature vol. xv* to have been nothing but *Mrs. Leigh's* trick.
Rise and Progress of the Brosterian System. London **1827.**

Bertrand, Alexandre Jacques Francois (1795-1831) was a French doctor whose fame rests on his work as doctor of psychology. He pronounced stuttering to be a spasmodic nervous affection, curable by any method which will distract the attention of the patient from his speech. He prophesied the downfall of the Leigh method as soon as it should have lost the secrecy which surrounded it and he took part in the French Academy discussion on Magendie's report, on this method.
Archiv. Gén. de Médecine. **1828.**

McCormac, Henry, was a doctor who announced a curative method for which he claimed infallibility. It consisted of using deep inspirations and forcible expirations, for he believed stuttering was caused by the attempt to speak with nearly emptied lungs.
The Cause and Cure of Hesitation of Speech, or Stammering. London, **1828.**

Arnott, Neil (1788-1874) a Scotch physician, attributed the cause to spasm of the glottis, and recommended the use of a continuous "e" sound between each word, to keep the glottis open.
Elements of Physics. Edinburgh, **1828.**

Müller, Johannes Peter (1801-1858) was a German physiologist and comparative anatomist who showed the dependence of physiology on other sciences, and expounded the mechanism of the voice, speech and hearing. He investigated the anatomy of the larynx. He, too, blamed spasm of the glottis, but advised the entire omission of explosive sounds in reading exercises, so as to keep the glottis open.
Handbuch der Physiologie des Menschen. **1833.**

Déleau, Nicholas (1797-1862) was a specialist in the maladies of the ear. He distinguished between the habit of vicious pronunciation, the stammering due to organic lesions, and three species of stuttering, one due to faulty tongue action, a second characterized by spasmodic closure of the lips, and a third in which spasm of the glottis takes place. The general cause of stuttering he held to be an infirm will, incomplete cerebral action, or deficient innervation. His treatment consisted wholly in educating the vocal and articulating organs, not by aid of the ear, but by sight, so as to secure their being placed in the right position for each sound enunciated.
Mémoire sur le Bégaiement—Revue Med. **1829.**

Palmer, Shirley, was a doctor who divided speech defects into two classes, organic and functional. The latter he held to be chiefly due to irregular muscular action which being established from habit becomes independent of the original cause and closely resembles chorea in its nature and phenomena. His treatment was one of moral discipline with varied details for each case.
Popular Illustrations of Medicine. London, **1829.**

---

*Vide infra.

Chègoin, H. de, believed the tongue to be of disproportionate size in stutterers, and attempted by mechanical methods to alter this disproportion.
Recherches sur les causes et le traitement du Bégaiement. Paris, **1830.**

Wutzer, P. This professor invented a tongue lever for the purpose of keeping the tongue out of the fossa behind the inferior incisors.
On Stuttering. Deutsche Klinik, **1850.**

Serre d'Alais recognized two species of stuttering, one a chorea of the muscles concerned in articulation, and the other a tetanus of the muscles of phonation and respiration. His method comprised the separate forcible pronunciation of every syllable aided by synchronous movements of the arms, in severe cases. He afterwards acknowledged its uselessness, according to a competitor.
Mémorial des Hopitaux du Midi, **1829.**

Magendie, F. (1783-1855) was a French physiologist and one of the creators of the school of experimental physiology. His greatest work was on the nervous system. He attributed speech to an organic intelligence, (instinct) and stuttering to a want of this intelligence. He believed that will power could replace this want sufficiently to bring about a cure in those possessed of enough moral force for its exercise.
Journ. Gén. de Méd. **1828.** *Dict. de Méd. et de Chirurg. Practique.* **1830.** *Précis de Physiologie.* **1833.**

Schulthess, Rudolf, compared stuttering to a hydrophobic spasm, and called it "phonophobia." He believed that spasm of the glottis extending through nerve associations to the other organs of speech was the main cause. He classified stuttering as idiopathic, due to want of harmony between nerve action and muscular action; symptomatic, and sympathetic, both due to other existing affections.
Das Stammeln und Stottern. Zurich. **1830.**

Bausmann was the professor appointed by the Prussian government to supervise the instruction of patients in the "Leigh method." He endeavored to improve on it by adding directions for the management of respiration, and for forming the initial letters of syllables.
Das Geheimniss stotternde und stammelnde zu heilen. Halle. **1832.**

Harnisch believed that the cause of the trouble was to be found in the larynx and the back of the tongue. He laid down rules for the government of the tongue and lips.
Preface to *Das Geheimniss Stotternde und Stammelnde zu heilen. Otto.* Halle. **1832.**

Otto located the remote cause in the mental condition of the stutterer, or in the debility of the nervous system, but the proximate cause was the condition of the speech organs. No general rule could be laid down, in his opinion, which would govern the treatment of all cases.
Das Geheimniss Stotternde und Stammelnde zu heilen. Halle. **1832.**

Bell, Charles, Sir (1774-1842) was a Scottish anatomist born in Edinburgh, who was responsible for the discovery of the different function of the nerves corresponding with their relation to different parts of the brain. He was professor of anatomy, physic and surgery, at the College of Surgery, London. According to him stuttering was due to chorea, depending upon an inadequate capacity properly to co-ordinate the different actions required for fluent speech, resulting from a certain debility of the nerves controlling the organs of speech. He noted the consequent disordered respiration, and stressed the supposed action of the larynx in articulation.
Philosophical Transaction. London. **1832.**

Poett, Joseph, held that the whole difficulty lay in the spasmodic action of the muscles of speech, as opposed to those concerned in phonation, and that this resulted from deranged nerve-function.
A Practical Treatise on Stammering. London. **1833.**

Cull, Richard, recommended systematic voice training by rhythmic means, under competent instructors, repeated until a correct sequence of speech-action is established in place of the faulty sequence.
Stammering and Its Cure. London. **1836.**

Berthold was a physiologist who defined stuttering as an impeded or unregulated motion of the organs of speech caused by faulty conditions of their muscular or nervous action. He thought that the muscles of the lower jaw might be affected.
Physiologie. Goettingen. **1837.**

Warren, Edward, held stuttering to be a depraved habit arising from an irregularity of function of the nervous system caused by weakness. Sometimes the organs of articulation are at fault, and sometimes the voice itself, and sometimes both; the result is great nervous agitation and contortion of the features.
Remarks on Stammering. Am. Journ. of Medical Sciences. **1837.** *The Cause and Cure of Stammering.*

Good, John Mason, maintained that stuttering should be treated by exercise of the organs on the difficult sounds, when the defect was of non-organic origin, and that the will should be strengthened to control the muscles of articulation.
Study of Medicine, Vol. I. London. **1840.**

Du Soit attributed stuttering to spasms of the respiratory system which take the form of tetanus or clonic spasm.
Gazette Médicale 2me série. Vol. viii. **1840.**

Hoffman, August Wilhelm (1818-1892) blamed spasm of the glottis, and advocated physical relaxation, and the avoidance of all muscular effort in the throat, tongue or lips, and avoidance of effort in other parts of the body as well.
Theoretische Praktische Anweisung zur Radikal-Heilung Stotternder. Berlin. **1840.**

Malebouche, M., purchased Mrs. Leigh's secret, and made a classification depending upon faulty tongue movements. He improved upon her method by advocating elevation of the whole tongue to the palate, and by directing treatment to the lips. He denied the existence of any but lingual consonants.
Précis sur les Causes du Bégaiement etc. Paris. **1841.**

Lee (1773-1877) was the English physician after whom is named the cervical ganglion. He believed that stuttering was a neurosis, which was intermittent, but that it had another form which was amenable to surgical treatment.

Dieffenbach, Johann Frederick (1795-1847) was a German surgeon who became famous as an operator, and later professor and director of clinical surgery in Berlin. He began the surgical treatment for speech defects which became fashionable in Europe for several years. His favorite operation was to make a horizontal section at the root of the tongue, excising a triangular wedge completely across and nearly through it, with the object of dividing the lingual muscles, and thereby interrupting their innervation, in order to modify or cure the muscular spasm. His method was the result of a fancied connection between defective articulation and strabismus.
Letter to the French Academy, March, **1841.** *Berliner Medizinische Central Zeitung.* **1841,** p. 163, etc.

Velpeau, Alfred Armand Louis Marie (1795-1867), professor of clinical surgery in Paris, member of the Institut, and writer and teacher of considerable influence, held that stuttering originated in an unusual depth of the palate, so he divided the hyo-glossi, genio-hyo-glossi, and stylo-glossi muscles, in different forms of the affection.
Annales de la Chirurgie Française etc. **1841.**

Amussat, Jean Zuléma (1796-1856) was a French surgeon and prosector at the Paris faculty of medicine, who published his research work on the nervous system. His treatment for stuttering consisted of dividing the genio-hyo-glossi muscles.
Letter to the French Academy. **1841.**

Baudens, Jean Baptiste Lucien (1804-1857) was a French surgeon connected with the hospital at Lille, and later with the military hospital in Paris. He operated in a manner similar to Velpeau.

Bonnet (1841) held stuttering to be primarily a nervous affection, but that secondarily it might be due to functional disorder of the organs of speech, which tended to persist after removal of the primary cause. He considered that all the phenomena were due to functional disorder of the tongue muscles, and operated to divide the genio-hyo-glossi.

Froriep, Robert (1804-1861) was a German physician who was for thirteen years Director of the Pathological Museum at the Charité in Berlin, and whose medical and surgical atlasses are well known. He made an important contribution to the electrical therapy of his day. He treated stuttering by dividing the genio-hyo-glossus on one side only.
Froriep's Notizen. **1841.**

Braid, James (1795-1860) was an English physician and surgeon who was especially noted for his researches on animal magnetism or neuro-hypnotism. He attempted to cure stuttering by excising the tonsils or uvula and was followed in this by various other English surgeons including *Yeardsley.*

Parker, Willard (1806-1866), professor of surgery in the College of Physicians and Surgeons in New York, was one of those who introduced the fashion of operative treatment for stuttering into America.

Mott, Valentine (1785-1865) was an American surgeon who studied medicine at Columbia University, London, and Edinburgh, who first introduced operation for the immobility of the lower jaw, also treated stuttering by operating.

Post, Alfred Charles (1806-1866) was an American surgeon who had studied also in Paris, Berlin and London. He too used the operative method for treatment of stuttering.
Observations on the Cure of Stammering. **1841.**

This fashion for curing stuttering by means of operation which was rampant in the early forties of the century varied considerably in its details. The German school in the main followed the practice of *Dieffenbach,* the French school that of *Velpeau,* and the English school, *Braid.* The results, however, though sometimes at first somewhat encouraging, soon proved to be of merely temporary duration, and when it was shown that not five per cent had received any permanent benefit, the method was hastily abandoned.

Hall–Marshall, (1790-1857) was an eminent English physiologist whose name is known in connection with the function of the reflex system. He held the essential cause of stuttering to be an imperfect act of volition due to the involuntary action of the reflex spinal center regulating articulation, which acted through irritation produced by excited incident nerves, the whole phenomena resembling a partial chorea. He refuted the theory that it could be spasm of the glottis but recommended the use of a continuous flowing manner of speech, avoiding as much as possible any interruption therein.
Diseases of the Nervous System. **1841.**

Wright, James, attributed stuttering to the faulty action of the velum palati and the organs used in the formation of the mute consonants.
Impediments of Speech. London. **1841.**

Colombat de l'Isère (*l. ca.* **1831)** borrowed the majority of his theories from predecessors, whose work he systematized. He divided stuttering into 1) Bégaiement labio choréique consisting of spasmodic action of the lips, tongue, etc.

2) Bégaiement gutturo-tetanique, consisting in rigidity of the muscles of the larynx and pharynx as well as those of respiration, producing spasm of the glottis, and characterized by sudden stoppage of the breath-current, and difficult enunciation of the gutteral sounds. He located the immediate cause in want of harmony between the nervous excitation which follows on thought and the muscular actions which are concerned in its expression. His treatment consisted in the use of rhythmic vocal gymnastics to educate the muscles of respiration, phonation and articulation to regularity of action, and by a system of opposing movements of the muscles of articulation he aimed at preventing the repetition of sounds. He also used many mechanical devices for the teeth and mouth, the most famous of which is his "muthonome." He was awarded the Monthyon prize from the French Academy for his efforts to cure stuttering.
Traité medico-chirurgical des maladies des organes de la voix. Paris. **1834.** *Du Bégaiement et tous les autres vices de la parole.* Paris. **1831, 1840, 1843.**

Jourdant in **1843** deposited a sealed envelope at the Academy of Paris containing a new system. *Becquerel* was authorized to publish it, and this led to *Becquerel's "Traité sur Bégaiement"* in which he recast *Jourdant's* views in more scientific form. The basis of this theory was that stammering expends the breath too quickly, therefore you must regulate the respiraton properly.

Beesel placed much stress on the position of the larynx in cases of stuttering, and recommended the exercising of the patient on the words which are found to be most difficult for him. He blamed also incorrect respiration.
Belehrung über die Entstehung, Verhütung und Heilung des Stotterns. Danzig **1843.**

Merkel, Carl Ludwig, who investigated the anatomy and physiology of the voice and speech considered that the primary cause of stuttering lies in an adynamic state of the organs, wholly due to a corresponding condition of the volition, resulting in a deeply-rooted habit of defective speech. According to him, treatment should be concentrated first upon an effort to raise the tone of the whole body, for lessening the force of the articulating organs, and for strengthening the energy of the respiratory function.
Encyclop. der Gesammten Medicin. **1844.** *Anatomie und Phys. des menschlichen Stimm-und Sprach-organs.* Leipzig **1857.** *Physiologie der menschlichen Sprache.* Leipzig **1866,** *Anthropophonik* **1870.**

Bühring held that interruption of the normal isochronism which should exist between the actions of the various nerve-tracts which influence the muscular apparatus of speech was the cause of stuttering and might be brought about in a variety of ways.
A Contribution to the Therapeutics of Stuttering. Caspar's Wochenschrift **1844.**

Lichtinger attributed stuttering to the predominance of the excito-motory (spinal) over the central (cerebral) nervous system, resulting either from the abnormal weakening of the latter, or from increased energy of the former.
Med. Zeitung No. 34. Berlin **1844.**

Blume, F., was a stutter-doctor who placed the proximate cause as want of harmony between thought, and speech. He believed that cases which exhibited the phenomena of the affection while singing or climbing a steep ascent to be incurable. In his treatment he used mechanical contrivances with exercise of the organs concerned. He was among those who for a time attempted operative treatment and is reputed to have performed forty such operations.
Neueste Heilmethode des Stotterübels. Leipzig. **1841.**

Hagemann, Mrs. Henrietta, blamed the tongue as the chief cause of stuttering and used the *Arnott* e-sound with the *Leigh- Yates* trick and the placing of an n-sound before each difficult syllable.
Untrügliche Heilung des Stotter und Stammel- Uebels. Breslau. **1845.**

Becquerel ascribed the proximate cause to faulty action of the respiratory muscles, which permitted an escape of air prematurely. He directed his treatment therefore towards obtaining the retention and controlled and economic use of the breath. He subsequently combined *Jourdant's* method with the gesticulatory method of *Serre d'Alais*.
Traité du Bégaiement. Paris. **1847.**

Graves, Robert James, (1797-1853) was an Irish physician famous as a clinician, lecturer and original investigator. He was one of the editors of the Dublin Hospital Reports and his book *Clinical Lectures on the Practice of Medicine* was used in every medical school in Europe. He adopted the spasm-of-the-glottis theory, to account for stuttering, and claimed to have discovered a method for its temporary relief, namely, to direct the attention of the patient away from his speech by having him strike some object synchronously with the words spoken, keeping strict time.
Clinical Lectures, edit. by Neligan. London. **1848.**

Comstock was a Philadelphia physician who taught a system of elocution with special reference to the alleviation of defects of speech. His method consisted in exercising the voice daily by reading aloud in unison with others.

Bishop, John F., considered that stuttering was most commonly induced by the attempt to speak without producing vibrations of the vocal cords. This was due to imperfect education or training of the organs of articulation, and deficiency in that sympathetic association which ought to exist between the articulatory and vocal organs.
On Articulate Sounds etc. London. **1851.**

Angermann, F., placed the primary cause as "defective volition of the mind upon the organs of speech," the resulting disturbance proceeding "either from the mind, from the central organs, from the nerves, or finally from the muscles," all the organs in speech being simultaneously seized by spasm. He was influenced by *Itard*.
Das Stottern, sein Wesen und seine Heilung. Berlin. **1853.**

Romberg, Moritz Heinrich (1795-1873) was a German pathologist and neuropathologist famous for his diagnostic description of ataxia. He placed stuttering among the neuroses of motility, under the subtitle of "vocal spasms."
Nervous Diseases of Man. London. **1858.**

Eich believed that the primary causes of stuttering were psychical rather than physical, and that abnormal respiration was the result, not the cause of stuttering. He denied that the stutterer ever tries to speak during an inspiration. He recommended treatment in an institution where each case could be studied separately, and treated according to its individual needs.
Die Heilung des Stotter-uebels und sonstiger Sprachfehler. Pesth. **1858.**

Hunt, James, treated stuttering successfully in England for many years, under the commendation of such observers as *Canon Kingsley*, and Mr. Liston the eminent surgeon. His treatment according to *Kingsley* was "naturally, and without dodge or trick, to teach the patient to speak consciously, as other men spoke unconsciously." By comparing the normal with the abnormal use of the organs of speech, he reduced the complicated phenomena to abuses of the action of the lip, tongue, jaw and breath, either singly or combined. His work was taken up successively by his son, *Dr. James Hunt*, of Hastings, and his son-in-law, the Rev. *H. F. Rivers*, of Tunbridge, who conducted an institute for the cure of speech defects and published an elaborate treatise on the subject. He described stuttering as in intermittent mania of the organs of speech, 22 varieties of which he differentiated and named. His treatment hinged upon disciplinary exercise of the organs of voice under the direction of an experienced instructor.
Stuttering and Stammering by James Hunt. 7th edit. London. **1870.**

Kingsley, Charles, Canon (1819-1875) the celebrated English orator and writer, and chaplain to the Queen, was himself a stutterer until he was 40 years of age. He believed that the cause of stuttering could be traced in three cases out of four to conscious or unconscious imitation. In a letter to a young lady he tells her that she stutters because her upper teeth, like his, shut over the lower ones, and prescribes a set of rules for her guidance in reading and speaking, the chief of which being to open her mouth widely, and read or speak from a full lung, breathing inwards at every stop, and keeping her tongue down. He advocated the use of dumbbell exercises to help breathing, and he also advised the placing of a bit of cork between the back teeth when speaking, keeping the upper lip drawn tightly down. He considered that childhood was an unsuitable time for treating the defect, and considered boxing as being "over and above a healing art" for the stammerer.
Irrational of Speech. Frazer's Mag. London. **1859.**

Leubuscher was professor of pathology at the University of Jena. The seat of spasmodic affections of the speech organs he believed to be located in the tracts of the hypo-glossal, facial and spinal accessory nerves. Its etiology he held to be frequently psychical, and recommended methodical speech-gymnastics to regulate the innervation—under the treatment of specialists.
Handbuch der Medicinischen Klinik. Leipzig. **1860.**

Rosenthal, M., professor of nervous diseases at Vienna, believed that the feeble development of the respiratory muscles during childhood was the cause of stuttering through the production of habitual disharmony of respiration and the lessening of the influence of the will. Later he placed the real pathogenic cause in an hereditary morbid excitability, a congenital weakness of the apparatus in the medulla oblongata governing respiration and phonation, which was affected in childhood by some physical shock. He described it as a disorder of the co-ordination concerned in the emission of sounds, increased by anxiety and embarrassment. He advocated a method of treatment based on rhythm, to be accomplished in special institutions.
Stottern. Weiner Medicinalische Wochenschrift. **1861.** *A Clinical Treatise on the Disorders of the Nervous System.* New York. **1879.**

Wolff, P. H., places the cause of stuttering either in the nerves or in the organs themselves, each case requiring careful examination in order to differentiate. He believed that it was a morbid disposition of the organs of speech rather than a disease, and classified it into three main varieties: 1) affecting the respiratory organs; 2) affecting the vocal organs; 3) affecting the articulatory organs, which could further be subdivided into five categories. His treatment consisted of trying all methods and almost all drugs, and finally of dividing the hypoglossal nerve.
Das Stottern und seine Heiling. Berlin. **1861.**

Violette considered stuttering to be a complex affection due to a single cause residing in the brain. His method consisted of vocal gymnastics, combined with gesticulation.
Etudes sur la Parole et ses défauts. Paris. **1862.**

Béclard, J., the physiologist, placed the real seat of the affection in the nervous system, and especially condemned surgical treatment.
Physiologie Humaine. Paris. **1862.**

Klencke, Hermann (1813-1881) was a German physician who practiced in Hanover and Leipzig and became later professor. He conducted an establishment for the treatment of stutterers. *Potter* states that he probably had the largest experience on the European continent in the treatment of stutterers, and speech defectives. His writings on the subject were prolific, and in the later ones he showed an inclination to undervalue the importance of any special theory in regard to treatment though he placed great stress on the influence of scrofula as a causative factor. He depended upon empirical methods derived from observation, experiment, and comparison of results in

many cases. He considered the regulation of diet and the adoption of hygienic methods to be of great importance, and he decried the use of drugs. He aimed at improving the nervous and muscular tone. He began his treatment by trying to arouse the will and keep it in constant action. Then he drilled the patient systematically in the technics of speech, a method which he described as "a medico-pedagogical system of speech-gymnastics." He doubted the ability of the ordinary physician to treat a case of stuttering as he considered that a special didactic method was necessary. He furthermore distinguished between stammering and stuttering, holding that the former had its causes in the articulating organs, and its manifestations in singing and declaiming as well as in talking, while the causes of the latter were rooted in the organs of respiration and phonation, with manifestation only in talking.
Die Störungen des menschlichen Stimm und Sprachorgans. Cassel **1844**. *Die Heilung des Stotterns.* Leipzig. **1862.**

Oré, J. R., considered stuttering as always a local affection, though primarily proceeding from a nervous cause. He advised the use of surgical measures for its cure, his objective point being the genio-hyo-glossi muscles.
Nouveau Dict. art. "*Bégaiement.*" Paris. **1866.**

Schulz, Erwin, believed that stuttering resulted from an augmented excito-motory influence of the spinal cord upon the muscles of respiration and articulation. His treatment was directed to diminish this influence and to strengthen that of the will. He believed that, by persevering in the use of voice-gymnastics, habitual stuttering might be cured.
Journal für Kinderkrankheiten. **1866.**

Chervin, M. Ainé, one of the most widely known and influential authorities in this field, was "Parish instructor" at Lyons in elocution and speech defects. He wrote about brain-irradiations, their comparative rapidity, continuity, energy and precision, and concluded that the higher brain was chiefly to blame for stuttering. He used many systems successfully, he claimed, but held that education of the will was most important. The co-ordinate power of the divers actions of the vocal organs must be strengthened by regular progressive language lessons, to teach the pupil to imitate the instructor's pronunciation.
Du Bégaiement considéré comme vice du Prononciation, Paris. **1867.** *Statistique du Bégaiement en France d'apres le nombre des conscrits bégues exemptés du service militaire de* **1850-1860.** Paris. **1878.**

Marshall, John, (1818-1891) was an English anatomist and surgeon, who became professor of anatomy at the Royal Academy of Surgeons. He blamed the irregular action of the nervous centers for stammering and stuttering.
Outlines of Physiology. London. **1867.**

Thomé held that stuttering was caused by the abnormal working of the central nervous system, induced by the influence of certain emotions which lead to respiratory disturbances. These fall into two groups: 1) Embarassment, uneasiness, and lack of confidence; 2) Excessive rapidity of thought, and an endeavor to produce the thought with corresponding rapidity.
Pathologie und Therapie des Stotterns. **1867.**

Lehwess, J., places the source of stuttering in either abnormal action of the respiratory organs, anomalies of individual muscles, abnormal psychical conditions, or conflict between the will and movements concerned in the production of speech. He believed rhythm to be a radical cure for the disorder.
Radicale Heilung des Stotterns. Berlin. **1868.**

Wyneken, C., held stuttering to be a neurosis, all the manifestations of which depend on the psychical condition of the patient, and that the larynx is the chief seat of the disturbance. The proximate psychical cause he held to be the defective influence of the will, produced by doubt. His treatment was

based largely on imbuing the patient with faith in his preceptor and in himself. He believed also in exercises for respiration, the voice and speech, accompanied by the rhythmical beating of time.
Uber das Stottern und dessen Heilung. Göttingen Zeitschrift für Rationelle Medicin. **1868.**

Coote, Holmes. A want of power to co-ordinate action, the source of which must be sought in the sensorium, was the cause of stuttering, in this doctor's opinion. Stammering, however, he believed to be due to timidity or to some temporary defect of the tongue.
Clinical Lecture. *British Med. Journal.* **1868.**

Guillaume, A., defined stuttering as an affection of irregularly intermittent type, having two characteristic symptoms: namely, the convulsive repetition of the same syllable and a convulsive stoppage at a syllable. He believed it to be due to defective co-ordination of the necessary muscular action. The foundations of his treatment lay in whispering exercises, for he believed that there was no essential difference in speech and whispering, other than the number and amplitude of the vibrations of the vocal cords. He also used lip gymnastics, and stressed the necessity of keeping the tongue up to the palate, and taking deep inspirations at every sentence.
Dictionnaire Encyclopédique des Sciences Médicales. art. "*Bégaiement.*" Paris. **1868.**

Kreutzer conducted a school in Rostock, Germany, which included instruction for the deaf and dumb, and stutterers and stammerers. He himself says that his method "includes not alone speech exercises, not alone gymnastics of the tongue, vocal organs, diaphragm and abdomen. . . rhythmical influence on the nervous system, technical instruction as to how any single sound must be spoken; it rather seeks to lay hold of the entire man, since not only are all of the above-named principles employed, but other means are also used according to the personality and character of the stutterer.

Brown-Séquard, Charles Edouard, (1817-1894), the French physiologist and neurologist responsible for the classification of the epilepsy called after him, and famous for his experiments on ameliorating debility, is reported to have stated verbally that in his opinion, stuttering should be considered as identical in its nature with the trembling of the eyelid or the lip.

Helmore held that stuttering was caused by neglect of muscular activity, and was the inability to regulate the action of the lips through weakness of the facial muscles, or inability to regulate the action of the tongue.
Speakers, Singers and Stammerers. London. **1874.**

Schrank considered stuttering to be a localized nervousness resulting in a compulsive act, the origin of which lies in the cortex. He believed besides, that it is always the consequence of mental inferiority.
Das Stotterübel. Munchen. **1877.**

Forbes, W. K., advocated the practice of the glottis stroke, the proceeding from an easy forcible whisper, to an easy forcible sound.
Elocution Simplified with Appendix on Lisping, Stammering, Stuttering, and other Defects of Speech. **1877.**

Kussmaul, Adolph, (1822-1902) of Graben, was a German physician, army surgeon and later professor at various German universities, whose outstanding work was on the pulse and respiration. He defined stuttering as a lalloneurosis, a "disarthria syllabaris", an intermittant spasmodic neurosis, in which there is a want of harmonius action between the muscles of respiration, vocalization and articulation, manifested by spasmodic articulation of syllables through spastic contractions of the articulating tube at its stop points for vowels and consonants, most frequently depending on irritability of the co-ordinating center which controls the formation of syllables, but may be induced by peripheral influences, due solely to a spasmodic inability to

execute that vocalization of consonants which is requisite for the formation of syllables, the attempt producing spasms of the muscles of speech which spreads to the phonic and respiratory centers. His treatment stressed education of the will power, breathing and articulatory exercises at first by vocal gymnastics directed to the pronunciation of separate and combined sounds, and later by rhythmic speech.
Disturbances of Speech, Ziemssen's Encyclopaedia of the Practice of Medicine. Vol. xiv. New York. **1877.**

Coen, of Vienna, was, according to Frōschels, the first doctor to specialize primarily in speech defects. He believed stuttering to be the result of deficient atmospheric pressure in the lungs caused by disturbances of innervation. Later he considered it to be due to pathological changes in the respiratory system which caused the breath spasm. His treatment was mainly along elocutionary lines, with Swedish exercises and electricity as accessories.
On the Heredity and Psychical Contagiousness of Stammering. Wein Med. Zeit. **1879.** *Spezielle Therapie des Stammelns.* Wein. **1899.**

Gunther, Edward, was principal of a German deaf-mute institute. He was author of a system of practical exercises for the cure of stuttering which aimed at regulating the respiration and bringing it under control of the will. The voice was trained first on the vowel sounds alone, then on the consonants and finally on the combined syllables, in a rhythmical manner, especial care being taken to avoid emphasizing the consonants.
Practical Instruction for the Cure of Stuttering., in the journal The Voice, Albany, N. Y. **1879.**

Werner, Edgar S., of Albany, who was editor of "The Voice," considered stuttering to be an affection of the vocalizing organs, and stammering one of the articulating organs. He recognized its varied etiology and treated these affections by elocutionary methods adapted to their individual necessities. His charge was $200. for each case. He did not profess any method peculiar to himself.
Stuttering and Its Causes. "The Voice." **1879.**

Howard, John, of New York, was a well known specialist in the treatment of defective speech. He held that stuttering was a throat contracting habit—a contraction of the glottis on vowel sounds, even when the stuttering appeared to be on consonants. Stuttering contracts the throat because the vocal organs do not receive a strong enough current from the lungs which are not sufficiently compressed or squeezed. He aimed at relaxing the constricted muscles of the chest and throat by what he termed "opposing movements." He charged $40 for a quarter of 20 lessons by mail, or $60 per quarter of regular office lessons.
See various articles in "The Voice." Albany. **1879-81.**

Bristowe, J. S., of St. Thomas' Hospital, London, defined stammering as a spasm of the vocal or articulating organs which might occur at the lips, the point, or the root of the tongue, or at the glottis; but whatever the part affected, the disorder consists "either in a sudden simple, more or less prolonged spasm; or in a series of such spasms in rapid sequence during which the implicated literal sound undergoes more or less frequent repetition," with generally a "tendency, especially if the spasm be prolonged for a greater or less number of other groups of muscles to be involved." He held the resemblances of different cases to each other to be far more important than their differences, and the pathological explanation to be the same in all. He was considered to be a high authority on nervous disorders in England. His suggested treatment included the possibility of the use of continuous galvanic current, attention to breathing, deliberate and carefully balanced enunciation, with practice on the most difficult combinations of syllables.
Voice and Speech, a Consideration of their Pathological Relations. Lumelian Lectures. **1879.**

Hammond, William Alexander, (1828-1900) was professor of nervous diseases in New York and gave his name to an athetosis. He ignored stuttering in his text book on nervous diseases, but wrote of the subject in journalistic publications. He called it a chorea of the muscles concerned in articulation, a defect of co-ordination, existing in two forms: 1) a difficulty of beginning the enunciation of sounds, especially those known as the explosives, which require the sudden opening of the lips; 2) that in which the sound is begun but is spasmodically reiterated. This latter form he calls stuttering. He considers it to be the result of a functional disorder of that part of the brain which presides over the faculty of speech, resulting in a want of the power of co-ordinating the requisite muscular action. He states that he cured himself of a severe stutter at the age of 19 by the use of a consentaneous muscular act with each troublesome word, such as tapping with the foot or the finger, for the purpose of attracting the attention from the effort to speak, thus rendering speech more automatic.
Stammering. The Voice, Albany, March, **1897.**

Shuldam, E. B., was a London doctor who classed stuttering and stammering together as being dependent on muscular spasm, originating in nervous weakness. This spasm leads to over-shallow breathing, which effects speech. He relied for treatment on elocutionary exercises of a rhythmical character, together with the regulation of respiration.
Stammering and its Rational Treatment. London. **1879.**

Gutzmann, A. Followed his father in his work with stutterers. A prolific research investigator; also a teacher of the deaf, who also made important contributions with regard to stuttering and the psychology of speech, drawing up rules for breathing and articulatrory exercises. Many M. D.'s and others from the United States used to work with him. Both his private clinic and the one at the Charité (Univ. of Berlin), continue under the direction of his son, Hermann Gutzmann—third generation. Hence their influence on the field over the world has been profound.
Sprachheilkunde. Das Stottern und seine grundliche Beseitigung. Berlin. **1879.**

Butterfield, L. A., was a professor of vocal physiology. He divided speech impediments into 1) minor defects; 2) stuttering, in which a sound is repeated again and again; 3) spasmodic hesitation; 4) convulsive stammering, in which the organs of speech are closed and held with intense pressure. The main proximate cause he believed to be a spasm of the diaphragm, which becomes automatic by habit. His treatment was purely in the nature of speech and respiratory gymnastics.
Stuttering and Stammering. The Voice. **1880.**

Bell, Alexander Melville, (1819-1905), who was born in Edinburgh and who was lecturer in Edinburgh University from 1843 to 1865, and later lecturer in the University of London, became instructor in Queen's College, Kingston, Canada, in 1870, coming finally to the United States where he founded a school of vocal physiology in Boston. He held that stuttering was simply a habit which could be uprooted at any stage by intelligent and persevering effort. He advocated for its treatment, reading aloud, in a strong, loud whisper, also counting while breathing in order to obtain a greater regularity of breathing.
Faults of Speech. Salem, Mass. **1880.** *Principles of Speech.* **1862.**

Potter, Samuel O. L., believed that the stuttering spasm implied deranged nerve function. He held that the spasmodic habit was set up and became automatic, and noted that it occurred at various stop points on the vocal tube, lips, tongue, larynx, or glottis.
Speech and Its Defects. **1882.**

Bates, Robert, was an American who treated stuttering with a variety of appliances: 1) a narrow flattened tube of silver applied to the median line of the roof of the mouth. This was used to aid the formation of the linguo palatal

letters. 2) A hollow bi-convex disc with a projecting silver tube placed between the lips to help labials and dental labials. 3) A belt and spring adjusted on the neck over the thyroid cartilage to help the gutterals.
See The Voice, Vol. vi. **1884.**

Findlay believed that the muscles of respiration were almost as much at fault as the organs of sound and articulation, and that though the action of the diaphragm in breathing and speaking is different, by the use of gestures the diaphragm may be made to take the same action as is necessary for making effective speech.
See The Voice, Vol. viii. **1885.**

Ruff held that the center of speech in stutterers has become accidentally exposed to some injurious influence *e. g.*, suffusion of blood owing to sudden expansion of the blood vessels, or insufficiency of the blood owing to their spasmodic contraction. The center of speech may then be said to be so disposed as to respond to any inducement however slight, to renew an attack of stammering. The first or provoking disturbance, arises from a lesion, shock, or some extraordinary, intense emotion.
Das Stottern, seine Ursachen und seine Heilung. **1885.**

Abbotts, W., believed the seat of stammering to be the larynx, and that of stuttering, which is a defective mode of expressing sounds, to be set more anteriorly.
Stammering, Stuttering and other Speech Affections. London. **1894.**

Payot followed a theory of educational therapeutics, based on educating the will.
L'Education de la Volunté. **1894.**

Sandow believed stammering to be a psychoneurosis based on the debility of the nerves involved in the action of speech, while each paroxysm of stammering was induced by psychic stimuli, caused either by dread of speaking, intense eagerness to speak, or by too violent innervation when talking. He notes that young children vocalize and articulate only when stimulated by feelings of ease and comfort. Fear and physical discomfort are antagonistic to this. Speech disturbances are due to injury to the speech nerves and impairment of the motor speech centers of the brain. Rest and relaxation are necessary.
Mekanik des Stotterns. Nordhaus. **1898.**

Thorpe, E. J. E., held that the muscles of the glottis tie up the passage because the energy is centered at some point above the diaphragm. The misplaced energy causes the spasm of the glottis. If it stops at the glottis, it affects the vowels only; if it extends to the lips and tongue it affects the consonants.
Speech Hesitation. New York. **1900.**

Rosenbach believed in an educational therapeutic method.
Nervöse Zustände und ihre psychische Behandlung. **1903.**

Dubois also followed a plan of educational therapeutics.
Les Psychonévroses et leur Ttraiement Moral. **1905.**

Rouma, G., advocated the use of arm gestures because cerebral activity actively overflows from the arm centers to the speech centers.
La Parole et les Troubles de la Parole. **1907.**

Behnke, Mrs. Emil, believed that the primary cause was inco-ordination of the breathing muscles with those of the vibrating element, which thus delayed production of tone. Laryngeal faults she held to be secondary.
On Stammering, Cleft Palate Speech and Lisping. **1907.**

Steckel called stuttering one of the worst forms of fear hysteria, and believed stuttering to be as much a psychological betrayal as are slips of the tongue and pen. The neurosis begins always in childhood, generally as a pure anxiety neurosis. He used psychoanalysis in treatment.
Nervöse Angstzustände und ihre Behandlung. **1908.**

Netskatschen was an advocate of the psychoanalytical method of treatment.
Eine neue psychologische Behandlungsmethode des Stotterns. **1909.**

Hudson-Makuen, G., was a Philadelphia laryngologist who believed that the primary cause of stuttering lay in the psychic realm but that treatment must aim at correcting the physical habits and drilling in the proper processes of speech.
The Laryngologist. Nov., **1910.**

Appelt, Alfred, also believed in psychoanalytic treatment for what he termed a neurosis.
Stammering and its Permanent Cure. **1911.**

Swift, Dr. Walter, holds that stuttering was visual central asthenia caused by absent or weak visualisation at the time of speech.
Journal of Abnormal Psychology, Vol. x. **1915.**

Coriat, Dr. Isador H., uses psychoanalysis for the treatment of stuttering. Insists that only an M. D. should be permitted to deal with the stutterer.
Stammering—a Psychoanalytic Interpretation." Journ. of Abnormal Psych. **1915.**

INDEX OF REFERENCES

Appelt, Alfred.....*Stammering and its Permanent Cure* (1911).
Bluemel, C. S.....*Stammering and Cognate Defects of Speech,* Vol. 2.
Fletcher, J. M.....*The Problem of Stuttering.*
Fröschels, Emil.....*Lehrbuch der Sprachheilkunde.*
Garrison.....*History of Medicine.*
Potter, S. O. L.....*Speech and Its Defects* (1882).
A. D. B.....*Allgemeine Deutsche Biographie.*
B. E.....*Biographie Universelle.*
E. B.....*Encyclopaedia Britannica.*
S. M. D.....*Steadman's Medical Dictionary.*
N. I. E.....*New International Encyclopaedia.*
G. E.....*Grande Encyclopédie.*

	Page
Abbotts—Bluemel	130
Aegineta—Potter, N. I. E.	116
Aetius—Potter, Garrison	116
Amman—Potter, N. I. E.	116
Amussat—Potter, N. I. E.	122
Angermann—Potter, Appelt	124
Appelt—Fletcher	131
Aristotle—Potter, Appelt, N.I.E.	115
Arnott—Potter	119
Astrié—Potter	118
Avicenna—Potter, N. I. E.	116
Bacon—Potter, E. B.	116
Bates—Bluemel	129
Baudens—Potter, N. I. E.	122
Bausmann—Potter	120
Béclard—Potter	125
Becquerel—Potter, Appelt, Fröschels	124
Beesel—Potter, Appelt	123
Behnke—Bluemel	130
Bell, A.M.—Potter, Bluemel, N.I.E.	129
Bell, C.—Potter, E. B.	120
Berthold—Potter	121
Bertrand—Potter, G. E.	119
Bishop—Potter, Bluemel	124
Blume—Potter, Fröschels	123
Bonnet—Potter	122
Braid—Potter, N. I. E.	122
Bristowe—Potter	128
Broster—Potter	119
Brown-Séquard—Potter, S. M. D.	127
Bühring—Potter	123
Butterfield—Potter	129
Catullus—Appelt, N. I. E.	115
Celsus—Potter, Garrison, Fletcher, N. I. E.	115

	Page
Charlier—Potter	118
Chègoin—Potter	120
Chervin—Potter	126
Cicero—Appelt, N. I. E.	115
Coen—Potter, Fröschels	128
Colombat—Potter, Appelt	122
Combe—Potter, N. I. E.	119
Comstock—Potter	124
Coote—Potter	127
Coriat—Appelt, Fletcher	131
Crichton—Potter	117
Cull—Potter	121
Cullen—Potter, N. I. E.	117
Darwin—Potter, N. I. E.	117
De Chauliac—Potter, N. I. E.	116
Deleau—Potter, G. E.	119
Delius—Potter	117
Demosthenes—Potter, Bluemel, Appelt, N. I. E.	115
Dieffenbach—Potter, Fröschels, N. I. E.	121
Du Soit—Appelt	121
Dubois—Appelt	130
Dupuytren—Appelt	118
Eich—Potter	124
Findlay—Bluemel	130
Forbes—Bluemel	127
Frank—Potter	118
Froriep—Potter, N. I. E.	122
Galen—Appelt, Fletcher, N. I. E.	116
Good—Potter	121
Graves—Potter, N. I. E.	124
Guillaume—Potter	127
Gunther—Potter	128
Gutzmann, A.—Appelt, Fröschels	129
Gutzmann, H.—Fröschels	129
Haen—Potter, A. D. B.	117
Hagemann—Potter, Fröschels	123
Hahn—Potter, A. D. B.	116
Hammond—Potter, S. M. D.	129
Harnisch—Potter	120
Hartley—Potter, N. I. E.	117
Helmore—Bluemel	127
Herodotus—Appelt, N. I. E.	115
Hildanus—Potter	116
Hippocrates—Potter, N. I. E.	115
Hoffman—Potter, N. I. E.	121
Howard—Potter, Bluemel	128
Hudson-Makuen—Fletcher	131
Hunt—Potter	124
Itard—Potter, B. U.	118
Jourdant—Appelt	123
Kingsley—Potter, Bluemel E. B.	125
Klencke—Potter, A. D. B.	125
Kreutzer—Potter	127
Kussmaul—Potter, Garrison, S. M. D.	127
Kustner—Potter	116
Lee—Potter, S. M. D.	121
Lehwess—Potter	126
Leigh—Potter	118
Lichtinger—Potter	123
Leubuscher—Potter	125
Magendie—Potter, B. U.	120
Malebouche—Potter	121
Marshall—Potter, E. B.	126
Marshall-Hall—Potter, E. B.	122
McCormac—Potter, Appelt	119
Menjot—Potter, N. I. E.	116
Mendlessohn—Potter, N. I. E.	117
Mercurialis—Potter, Garrison	116
Merkel—Potter, Garrison, Fröschels	123
Morgagni—Potter, N. I. E.	117
Mott—Potter, N. I. E.	122
Müller—Potter, Appelt, N. I. E.	119
Netskatschen—Fletcher	131
Oré—Potter	126
Otto—Potter	120
Palmer—Potter	119
Parker—Potter, N. I. E.	122
Payot—Appelt	130
Poett—Potter	120
Post—Potter, N. I. E.	122
Potter—Bluemel	129
Romberg—Potter, N. I. E.	124
Rosenbach—Appelt	130
Rosenthal—Potter, Fröschels	125
Rouma—Bluemel	130
Ruff—Appelt, Fröschels	130
Rullier—Potter, Appelt	118
Sandow—Appelt, Bluemel	130
Santorini—Potter, Garrison, N. I. E.	117
Satyrus—Potter, Bluemel	115
Sauvages—Potter	117
Savary—Potter	118
Schrank—Potter, Fröschels	127
Schulthess—Potter	120
Schulz—Potter	126
Serre d'Alais—Potter	120
Shuldam—Potter, Bluemel, Fröschels	129
Steckel—Fletcher	130
Swift—Fletcher	131
Thelwall—Potter	118
Thomé—Appelt	126
Thorpe—Bluemel	130
Valerius Maximus—Appelt	115
Velpeau—Potter, N. I. E.	121
Violette—Potter	125
Voisin—Potter	118
Warren—Potter	121
Watson—Potter	117
Werner—Potter	128
Wolff—Potter	125
Wright—Potter	122
Wutzer—Potter	120
Wyneken—Potter	126
Yates—Potter, Bluemel	118
Zitterland—Potter	118

RESPIRATORY CARDIAC ARRHYTHMIA IN STUTTERING*

MARTIN F. PALMER, M.A., Sc.D.
Director, Flo Brown Memorial Laboratory,
University of Wichita

ANNA MAE GILLETT, A. B.
Assistant in Charge of Research,
Flo Brown Memorial Laboratory,
University of Wichita

As in a previous paper on the cardiac rhythms of stutterers,[1] this investigation represents no attempt to determine in toto the causation of stuttering, but offers certain laboratory evidence that must be taken into consideration in further etiological research.

Sinus or respiratory arrhythmia is of common occurrence in children and young adults. In its usual pattern, it consists of an inspiratory decrease in the length of the heart beat, and an expiratory increase in the length of the heart beat. According to Lamson, it is most frequently found in "nervous individuals.[2] It is more marked in girls than in boys, and while less sinus arrhythmia is found among the adult population, in those individuals who have it there is a tendency for it to increase as they grow older.[3] Surprisingly enough, although it is linked definitely to the respiratory rhythms of the individual, it is actually not caused by the respiratory rhythms per se, since in dogs, destruction of the respiratory neural pathways does not affect the respiratory arrhythmia, which continues to fluctuate as though the respiratory mechanisms were intact.[4]

Lamson further remarks that respiratory cardiac arrhythmia is a normal condition and shows an active respiratory center.[5]

Since it has been shown in studies from this laboratory and others that the cardiac rhythms of stutterers are more irregular than their normal controls when neither are speaking, in other words, that this is a constant functional difference between stutterers and

---

*Presented by Martin F. Palmer to the American Speech Correction Association Convention at Cleveland, Ohio, December 28, 1938.

[1]Palmer, Martin F. and Gillett, Anna Mae. Sex Differences in the Cardiac Rhythms of Stutterers. *Journal of Speech Disorders*, March, 1938.

[2]Lamson, Paul Dudley. The Heart Rhythms. Williams and Wilkins Co. 1925.

[3]Lincoln, Edith M. The Hearts of Normal Children. *Am. Jour. Dis. Children*, 35 (6), 1001–1020. 1928.

[4]Heymans, J. F. et C. Sur le mécanisme de l'arhythmie cardiaque respiratoire. Compt. Rend Soc. Biol. 96 (10): 716–718. 1927.

[5]Lamson, Paul Dudley. (*ibid.*)

normals not directly associated with the stuttering complex, it would be expected that stutterers would show more respiratory arrhythmia than normal speakers. Although Travis, Tuttle, and Cowan[6] reported a few cases of sinus arrhythmia in their stutterers, and the same is confirmed in this paper, such is the exact reverse of the actual situation for most stutterers.

The present study compares the occurrence of respiratory cardiac arrhythmia in stutterers and normal speakers when neither are speaking. The subjects were twenty-one stutterers and twenty-four normal speakers, with an age range of from 9–26 years. Each stutterer was matched age for age and sex for sex with at least one normal control.

As with the previously reported experiment, the study was carried out for the most part in an isolated, sound-proofed room without windows. Records were run approximately two hours after meals in order to standardize the change in cardiac rhythms caused by the digestive processes.

It is important to note that in this investigation, neither normals nor stutterers were speaking, and that both sets of subjects knew they were not going to have to speak. Both sets of subjects were seated so that the movements of the styli of the apparatus were not visible to them, and the results quoted later can be said to be the result of constant differences between stutterers and normals aside from the speech defect directly. This is not a study of the direct stuttering symptomatology. Thus these results are simply evidence of a deep-rooted neuro-physiological difference between stutterers and normals.

The records were taken on a Miller kymograph. Lombard pneumographs were utilized for the respiratory excursions of the thorax and abdomen, and the radial pulse was recorded by means of the Lombard radial pulse apparatus. The records were timed by an electromagnetic timer operating at .20 second. This type of apparatus was chosen since it is not only convenient to use, but is highly accurate for qualitative measurements. Also the subjects undergo a minimum of discomfort, and no training is required of the subjects before a record can be run.

The records were analyzed by the Lombard technics,[7] insuring accuracy to the second decimal.

A note must be taken in regard to the radial pulse. Although it represents the duration of the cardiac cycle very precisely, it should be remembered that the occurrence of radial systole is delayed approximately .10 second from the occurrence of aortic systole. This error will displace the heart beat occurring at the onset of each inspiratory or expiratory movement. Since this counterbalances in the case of respiratory arrhythmia about one-sixth to one-eighth of the heart beats counted, displacing them into inspiration or

[6]Travis, L. E., Tuttle, W. W. and Cowan, Donald W. A Study of the Heart Rate During Stuttering. *Journal Speech Disorders*, March, 1936.

[7]Lombard, Warren P. Laboratory Work in Physiology. 3d ed. Geo. Wahr. 1920. pp. 276–284.

expiration as the case may be, the practical result in this investigation is to decrease the mathematical significance of the results. However, the results are so clear from the data at hand, and also since the error appears likewise in the stuttering data, the error has not been corrected for these results. Any change which would occur in this data from a calibration of the .10 second delay would only increase the mathematical significance of the results.

Stutterers as a group differ from normals as a group in the following manner (Table I): During inspiration significantly fewer of the stutterers' heart beats decrease in length than the normals, and significantly more of the stutterers' heart beats remain at the same rhythm than the normals. During expiration, significantly fewer of the stutterers' heart beats increase in length than the normals, and significantly more of the stutterers' heart beats decrease in length than the normals. Stutterers may then be said to be in so far as the mechanism of respiratory cardiac arrhythmia is concerned, directly opposite in their fundamental physiological tendencies to normals.

TABLE I

	Normals		Sigma		3 Sigma	Stutterers
(Expiration)						
Increase	715	51.25%	18.67—1.34%		4.02%	#45.99%[8]
Decrease	422	30.25%	17.16—1.23%		3.69%	#36.76%
No change	231	16.56%	13.88—0.99%		2.97%	15.51%
Inc.-Dec.	27	1.94%	5.15	0.37%	1.11%	1.74%[9]
(Inspiration)						
Increase	295	20.97%	15.27	1.09%	3.27%	18.75%
Decrease	681	48.40%	18.75	1.33%	3.99%	#44.40%
No change	425	30.20%	17.22	1.22%	3.66%	#37.07%
Inc.-Dec.	6	00.43%	2.44	0.17%	0.51%	0.08%

The interesting conclusion can be drawn that during expiration, which is of course the motor portion for speech, fewer stutterers have respiratory arrhythmiac tendencies, but that when they do, these tendencies are directly opposed to the normal tendency. The note is worth taking that stuttering may possibly be caused from more than one physiological etiology. There is much laboratory and clinical evidence to this effect.

Since it was shown in a previous paper that the etiology of stuttering, so far as the heart rhythms are concerned, is definitely sex differentiated it next becomes necessary to discuss the differences found in these results.

When female normals are balanced against male normals certain interesting facts emerge. In the first place, the picture of increase

[8]Those differences which fall outside of a t score of 3.00 are starred. These are mathematically significant.

[9]It will be noted that in every table there is a line of figures headed Inc.-Dec. Here the heart beat both increased and decreased during the respiratory movement. These are irregularities of the pulse from stimulus, mental excitement, etc. It is a good indication of the constant condition of the experiment that the number is so low. It will be noted that there is no difference between the results for the stutterers and the normals.

in length of heart beat during expiration is found largely in the female sex. Males have a significantly lower ratio of increase than females. At the same time they have a larger ratio of decrease in the length of heart beat. Further, a larger ratio of them show signs of no respiratory cardiac arrhythmia. This confirms definitely Lincoln's study, earlier quoted. Thus respiratory cardiac arrhythmia is definitely linked to the female sex. Upon examining this condition in stutterers, it is seen that no difference exists between the sexes when they are stuttering. Both show the same tendencies in regard to respiratory arrhythmia during expiration and this distribution is the same for the male normal speakers. The conclusion seems inescapable that stuttering is a sex-linked metabolic condition.

TABLE II

(Expiration—Normals)

♀			Sigma	3 Sigma	♂
Increase	275	65.01%	2.32%	6.96%	#45.27%
Decrease	105	24.82%	2.10%	6.30%	#32.61%
No change	31	7.33%	1.02%	3.06%	#20.58%
Inc.-Dec	12	2.84%	0.81%	2.43%	1.54%
(Expiration—Stutterers)					
Increase	154	47.53%	2.76%	8.28%	45.39%
Decrease	112	34.57%	2.64%	7.92%	37.62%
No change	51	15.74%	2.01%	6.03%	15.41%
Inc.-Dec	7	2.16%	0.81%	2.43%	1.58%

Turning to inspiration and making the same comparisons, the same results occur. The responsibility for respiratory cardiac arrhythmia is found to be largely in the female sex. A significantly higher ratio of increase in length of heart beat is found in the males. A significantly lower ratio of decrease is found in the males, while a larger proportion of the males' heart beats do not change at all in rate during inspiration. In turning to the stutterers' results it is seen that when either sex is stuttering, no differences in regard to respiratory cardiac arrhythmia appear, but both sexes resemble the results found in male normals. The conclusion again must be that maleness and stuttering are linked not only as a chance proposition, but for definite physiological reasons. (Table III.)

TABLE III

(Inspiration—Normals)

♀			Sigma	3 Sigma	♂
Increase	68	15.92%	1.77%	5.31%	#23.16%
Decrease	276	64.64%	2.31%	6.93%	#41.32%
No change	81	18.97%	1.90%	5.70%	#35.10%
Inc.-Dec	2	0.47%	0.33%	0.99%	0.41%
(Inspiration—Stutterers)					
Increase	48	14.77%	1.97%	5.91%	19.88%
Decrease	154	47.38%	2.77%	8.31%	43.23%
No change	123	37.85%	2.69%	8.07%	36.77%
Inc.-Dec	0	00	00	00	0.12%

It becomes necessary next to compare female stutterers to female normals, and male stutterers to male normals. During expiration it is observed as would be expected from the above that female

stutterers have less increase in ratio of length of heart beat, more decrease in ratio of length of heart beat, and less tendency towards cardiac respiratory arrhythmia than their controls. Male stutterers are, however, a little different from this picture. There is no significant difference in the number of heart beats which increase in length from their controls, but significantly more heart beats decrease in length during expiration. Also in the male sex, more stutterers (male) have less tendency towards respiratory cardiac arrhythmia than their normal controls. (Table IV.)

TABLE IV

Normals			Sigma	3 Sigma	Stutterers
(Expiration—♀)					
Increase	275	65.01%	2.32%	6.96%	#47.53%
Decrease	105	24.82%	2.10%	6.30%	#34.57%
No change	31	7.33%	1.02%	3.06%	#15.74%
Inc.-Dec	12	2.84%	0.81%	2.43%	2.16%
(Expiration—♂)					
Increase	440	45.27%	1.60%	4.80%	45.39%
Decrease	317	32.61%	1.50%	4.50%	#37.62%
No change	200	20.58%	1.30%	3.90%	#15.41%
Inc.-Dec	15	1.54%	0.40%	1.20%	1.58%

During inspiration female stutterers are sharply differentiated from their controls. Their heart beats do not decrease in length as frequently as do their controls, and a much higher proportion show no tendency toward respiratory arrhythmia. Peculiarly, the male in inspiration show no significant differences from their normal controls. (Table V.) The conclusion again seems inescapable that while respiratory arrhythmia or lack of it may not be the cause of stuttering, it is linked to this sex-linked mechanism that causes stuttering, and the mechanism is of neuro-physiological order.

TABLE V

Normals			Sigma	3 Sigma	Stutterers
(Inspiration—♀)					
Increase	68	15.92%	1.77%	5.31%	14.77%
Decrease	276	64.64%	2.31%	6.93%	#47.38%
No change	81	18.97%	1.90%	5.70%	#37.85%
Inc.-Dec	2	0.47%	0.33%	0.99%	0.70%
(Inspiration—♂)					
Increase	227	23.16%	1.35%	4.05%	19.88%
Decrease	405	41.32%	1.57%	4.71%	43.23%
No change	344	35.10%	1.52%	4.56%	36.77%
Inc.-Dec	4	0.41%	0.20%	0.60%	0.12%

It was observed above that the picture when either sex is stuttering looks very much like the patterns presented by the male normals. However, there are differences which are significant when the female stutterers are opposed to the male normals. During inspiration, the female stutterers have significantly lower ratios of increases in the length of heart beat than male normals, and higher ratios than the males in the number of heart beats which decrease in length. This is an approximation, or a tendency in the female direction.

Thus stuttering in the female sex is somewhat different in its appearance in regard to respiratory arrhythmia than in the male sex. It seems clear that a differing mechanism is involved. The conclusion would seem acceptable that females are somewhat resistant to those factors which cause stuttering, and changes in the normal cardiac-respiratory relationship. During expiration, however, the female stutterers show more cardiac arrhythmia than do the male normals. Thus cardiac arrhythmia increase or decrease is not the cause of stuttering, but can be considered to be part of a direct by effect of a sex linked neuro-physiological-metabolic mechanism causing the stuttering, which cause is probably not the same from individual to individual.

TABLE VI

(Comparison of Mean Time Change—Expiration)

	Mean	Diff.	σ Diff.	$\frac{\text{Diff.}}{\sigma \text{ Diff.}}$
Normals	+.062 sec.	.033	.0085	3.88#
Stutterers	+.029 sec.			
♀ Normals	+.122 sec.			
♀ Stutterers	+.041 sec.	.081	.0150	5.40#
♂ Normals	+.035 sec.			
♂ Stutterers	+.025 sec.	.010	.0095	1.05

The foregoing differences are the result of a detailed qualitative study which considered only the fact that pulse rates increased, decreased or did not change during respiratory movements. A quantitative study yields further interesting information.

This comparison consists of examining the differences first of all in mean time change of the heart beat during respiratory excursions.

During expiration both normals and stutterers show a gain in the length of the heart beat, when all increases and losses are averaged. Normals, however, gain much more than stutterers, that is, the heart beat slows down more. When female normals are opposed to female stutterers, and male normals are opposed to male stutterers it is seen that this increase in the length of the heart beat is primarily in the female sex, while the male stutterers show no difference in this regard between themselves and male normals. (Table VI.)

During inspiration, both stutterers and normals show a decrease in the length of the heart beat, when all results are averaged. Female stutterers show significantly less increase than female normals. No difference is found in the male sex, whether normal or stuttering. In this regard, females differ from males when stuttering, and it is seen that although it is normal for females to show a high preponderance of quantitative cardiac respiratory arrhythmia, when they stutter they approach more closely the effects seen in the male sex. (Table VII.)

These increases or decreases in the length of the heart beat may be judged quantitatively in still a further way. The question arises whether this gain or loss is a steady one, or whether, since it has been shown qualitatively that this is not a constant phenomenon,

these changes occur more or less regularly in the types of subjects involved.

The best statistical measure of the regularity of occurrence is the standard deviation.

TABLE VII

(Comparison of Mean Time Change—Inspiration)

	Mean	Diff.	σ Diff.	Diff. / σ Diff.
Normals	—.040 sec.			
Stutterers	—.032 sec.	.008	.0051	1.57
♀ Normals	—.077 sec.			
♀ Stutterers	—.030 sec.	.047	.0083	5.66#
♂ Normals	—.023 sec.			
♂ Stutterers	—.034 sec.	.011	.0063	1.75

When expiration is examined it is noted that normals and stutterers classed as a whole do not differ from each other in the regularity at which the heart gains or loses speed during respiratory movements. That this is false is easily seen when the sexes are separated, since the one result counterbalances the other. Males and females when stuttering are directly opposed to each other in regard to regularity of this gain or loss. Female normals are much more irregular in this increase than female stutterers, while male normals are much more regular than male stutterers. (Table VIII.)

TABLE VIII

(Comparison of the Standard Deviations)

	σ	Diff.	σ Diff.	Diff. / σ Diff.
Normals	.216			
Stutterers	.209	.007	.0060	1.17
♀ Normals	.266			
♀ Stutterers	.189	.077	.0117	6.58
♂ Normals	.184			
♂ Stutterers	.216	.032	.0068	4.71

Again, upon looking at inspiration, it is seen that the mechanism is slightly different. Here all normals have less regular rates of increase and decrease, as do female stutterers and male stutterers opposed to their normal controls. (Table IX.)

TABLE IX

(Comparison of Standard Deviations—Inspiration)

	σ	Diff.	σ Diff.	Diff. / σ Diff.
Normals	.140	.022	.0035	6.29#
Stutterers	.118			
♀ Normals	.135			
♀ Stutterers	.092	.043	.0059	7.29#
♂ Normals	.140			
♂ Stutterers	.126	.014	.0044	3.18#

These results lead to the question of further etiological research in stuttering. Respiratory cardiac arrhythmia has been shown to be dependent not on the respiratory movements primarily, but rather as an essentially reflex phenomenon controlled by the state of the somatic circulation.[10] It has been produced experimentally by the injection of calcium gluconate solution.[11] The question of its production or inhibition by mental excitement does not enter into this picture since the differences in the sex index play so prominent a role. It would seem that the most favorable realm for research would lie in the bio-chemistry of the blood stream, and in the study of the various nutritive conditions which make for changes in the blood stream. The effect of the gonadal hormones on the blood stream seems an especially fruitful field. Further, since laterality has been shown to be sex-linked[12] and also dependent upon vitamin B nutrition, which in itself is linked to all of these problems, it would seem that further investigation along the lines of the cerebral gradient theory should not be overlooked.

General Conclusions

1. Qualitatively, stutterers differ from normals in regard to the respiratory increase and decrease of the length of their heart beats.

(a) The mechanisms of inspiration and expiration are not excatly the same, but stutterers can be said in general to have exactly opposite tendencies from normals.

2. All stuttering may not be caused by a single etiology.

3. Respiratory cardiac arrhythmia is primarily female in normals, but when females stutter they do not have as great a tendency to respiratory arrhythmia.

(a) Females stuttering differentiate sharply from their female normal controls, and approach the pattern of the male.

(b) Males, however, also differ from their normal controls, but in not the same way as females. During inspiration, they do not differ from their controls.

4. A quantitative study of the mean and standard deviations of the time involved from one cardiac systole to the next shows that the two sexes differ in this regard when they stutter, and further that stutterers increase their heart beats on inspiration much more regularly than the normals. However, on inspiration, female stutterers are more regular than their normal controls, while male stutterers are more irregular.

5. A differing mechanism is involved between the two sexes in stuttering.

6. Stuttering is the result of a sex-linked, neuro-physiologico-metabolic etiology.

---

[10]Heymans, J. F. et C. (*Ibid.*)

[11]Berliner, Kurt. The Effect of calcium injections on the human heart. *Am. Jour. Med. Sci.* 191 (1): 117–121. 1936.

[12]Travis, L. E. Speech Pathology.

EAR TRAINING IN THE TREATMENT OF ARTICULATION DISORDERS

C. VAN RIPER,
Western State Teachers College,
Michigan

Many texts in speech correction agree that the first step in remedial treatment of articulatory cases should be ear training, and most speech correctionists employ it. The exact nature of this ear training is too often vague, unsystematic, and perfunctory, although it is probably the most important tool in the clinician's kit. If the preliminary ear training is done well, little difficulty is experienced, even with the most severe cases. The speech correction teacher is prone to slight it because immediate results are not forthcoming, because it demands strong motivation, because it necessitates lesson preparation and clever techniques, and because she does not realize its nature or importance. Many parents and teachers feel that all they need to do to get rid of such a speech defect is to tell the child that he has said the word wrongly and must try it again. Often they attempt to show him by increasing the loudness of the correct word as though he were hard of hearing, a proceeding which is obviously poor pedagogy. It may be said with the utmost emphasis that no teacher should attempt to get a child to make a new speech sound without having first given him systematic ear training.

What are the aims and methods of this preliminary ear training? The aims are as follows:

1. *The child must be convinced that he has a problem which he must solve*, and this is not as easily done as may seem to be indicated. Due to sheltered environments and the tolerance of associates who have grown up with the speech difference, many speech defectives grow to adulthood without ever having been made aware of their speech disorder, which may be so noticeable as to shriek its presence whenever its possessor opens his mouth. If friends and acquaintances will not mention it, certainly the average stranger will not. We seldom hear ourselves speak. Instead, we listen to our vocalized thinking. And so the speech defective himself has little chance of becoming fully aware of the nature or frequency of his errors.

2. *The correct sound and the error must be isolated from their contexts.* As we have pointed out before, as long as a sound is lost within a word it cannot be heard or felt with any clarity. The word configurations must be broken up, so that the correct sound and the error can be seen by themselves.

3. *The correct sound and the error, each by itself, must come to have an individuality and an identity.* Many people fail to realize that before we can have discrimination we must have identification. All good teachers of speech correction give personalities to the sounds with which they work. They give them names, traits, even faces. From such identification comes recognition; from recognition comes discrimination; from discrimination comes success.

4. *The child must be strongly stimulated with the correct sound.* The child must hear the correct sound as he has never heard it before. He must hear it everywhere speech is spoken. It must ring in his ears, and every agency available should be used to provide this stimulation.

5. *The child must be given repeated opportunities to achieve a very clear discrimination between correct and incorrect sounds.* In one sense, all preceding four steps are pointed at facilitating this auditory discrimintaion, for it is the essence of the necessary ear-training of which we hear so much. Without the ability to differentiate correct sound from error, the student becomes discouraged, the treatment becomes blind drill, and the teacher wishes she had taken up library work.

STUDIES IN THE PSYCHOLOGY OF STUTTERING, XI.

Stuttering in Relation to Position in the Family*

JULIAN B. ROTTER
University of Iowa

This study considers certain factors of possible etiological significance in relation to stuttering. It is of particular interest that these factors can not be regarded as having any basis in heredity, and they can not be regarded, therefore, as representative of the so-called innate constitutional neuromuscular condition of the stutterer. Moreover, the factors considered stand in an asymmetrical relation to stuttering, if related to it at all, in the sense that they can hardly be effects, rather than causes, of the disorder. (Whether or not they are causes is another question.)

These factors are: (1) position in the family, such as oldest, youngest, only child, etc., (2) the number of years between the stutterer and his nearest sibling, and (3) the sex of the nearest siblings.

A careful review of the literature concerning the importance of position in the family shows that this aspect of the child's early environment is definitely related to the development of behavior patterns (1, 9, 10, 11, 12, 16).

Studies on delinquency, maladjustment, school achievement, psychoneuroses, and psychoses have shown the only child to be particularly handicapped in making a normal adjustment (2, 4, 9, 12, 14, 16, 17). Other studies show the oldest and youngest child most likely to have difficulty in adjusting as compared to middle children (2, 9, 13, 14, 17).

A few studies seem to indicate the contrary. Fenton (6) studied the behavior traits of only children in school and concluded that they did not show behavior significantly different from that of other children. Fenton's only criteria were teachers' ratings. In view of the wide differences that have been shown, in recent work (12), between the judgments of teachers as compared to those of clinical psychologists and psychiatrists and in view of the fact, as pointed out by Adler (2), that only children, although badly adjusted in their relations with other children, often become "teachers' pets," this study cannot be taken as conclusive. Hardy (7), in studying

*All data and materials pertinent to this study are on file in the State University of Iowa, Department of Psychology. The study was done under the direction of Wendell Johnson.

position in the family in relation to maladjustment in school, states in her discussions that position does not play an important role. Analysis of her data, however, shows that, when equated for socio-economic status, only 3.0 per cent of her well-adjusted group are only children as compared to 10.3 per cent of her maladjusted group.

That the number of years between children is important in understanding the personality of the child has been pointed out by Adler (3). However, this factor has not been considered in most of the research done on position in the family. It can be seen that this is in reality a special case of position in the family; that is, oldest and youngest children separated from their siblings by five or more years may be considered psychologically as only children, in several respects. Likewise, middle children separated from their siblings by five or more years, on one side, may be psychologically similar to oldest or youngest children in their training.

Busemann (5) found that the highest average ranks in school achievement were obtained by children in sexually homogeneous sibships. Scholarship was found to diminish progressively according to the number of opposite sexed siblings in a child's family. Hardy (7) reports that in her group of school children who were considered to be maladjusted, the nearest siblings of the maladjusted child were more frequently of the opposite sex. Thurstone and Jenkins (16) and Levy (11) in studying delinquents and problem children found that the nearest siblings to their subjects were more often of the same sex.

The most recent and comprehensive study on position in the family was done by Krout (10). Krout controlled the number of children in the family and the sex of the surrounding children, listing 26 ordinal types. These 26 types were studied with reference to 38 behavioral factors. Two findings of interest in connection with the present study were that the degree to which a particular child was favored by his parents was in inverse proportion to the number of children of the same sex as his own in the family, and that male children tended to be favored more than female children by the mother. When we consider that, in general, children spend most of the day with their mothers we might conclude that male children tend to be pampered more than female children. If this hypothesis were true it might be an important factor in the explanation of sex differences in stuttering. Krout concludes his study by stating, "A brief inspection of graphs constructed for each ordinal position discloses that each curve represents a constellation which is distinctively different from every other."

Louttit (12), Thurstone and Jenkins (16), and Jones (9), give summaries of some of the work done on position in the family.

Subjects

Five hundred and twenty-two cases of stuttering were analyzed. Of these 425 were male and 97 female. The age range was from 2.5 to 44 years. Only 18 of these cases were below five years of age.

The median age was 14 years and the distribution gave a bimodal curve with modes at 10 and 18 years. These cases represented present cases at various clinics in the mid-western area[1] plus 226 cases from the files of the University of Iowa clinic.

Comparable data regarding position in the family, for a presumably non-stuttering population, were taken from the report of the White House Conference on Child Health and Protection (18). These norms were computed in terms of 7,738 children of junior high school age. These norms, although placing the number of only children higher than any other set of norms in the literature, seemed to be highly reliable. As the average age of these children was about thirteen years, some of the families may not have been completed; however, this was also true of about half of our own group. A weighting of city children and the inclusion of 751 cases of delinquency may have influenced the percentages in the direction of a greater percentage of only, oldest and youngest children.

In analyzing the number of years between siblings, norms were computed in terms of the number of years between any two non-stuttering siblings of a stutterer. In this manner the same families were used and socio-economic status was controlled.

Results

The percentage of children who were only, oldest, youngest or middle children was computed for the stuttering group. These percentages were compared to the corresponding percentages for the non-stutterers, as taken from the White House Conference report. Statistically significantly more only children, and fewer middle children were found among the stutterers than could be expected by chance. (Table I.)

This order of more only children and fewer middle children, than can be accounted for by chance, is the order Adler (3) and Rosenow (13) would give as that in which we would expect "maladjustment" to occur. This order can best be looked on as that in which we would expect the child to be pampered, all other things being equal. That is, in general, only children are pampered more than oldest, youngest or middle children, and middle children are pampered less than only, oldest or youngest children.

The term pampering is used here in the broad sense. It would include the types of behavior which are sometimes called spoiling, babying, favoring, over-protecting, etc. It can be described as that type of training which affects the child so that he becomes dependent

[1]Data were also obtained from: Arlien Sinn, Eau Claire, Wisconsin, public schools; Melba Kimmell, Marinette, Wisconsin, public schools; Jane Shover, East Chicago, Indiana, public schools; Alonzo Morley, Brigham Young University; Jeanne Kellenberger, Racine, Wisconsin, public schools; Spencer Brown, Ohio State University; Esther Glaspey, Rochester, Minnesota, public schools; George Egland, University of Minnesota; Bryng Bryngelson, University of Minnesota; H. E. Scarbrough, University of Iowa; Enod House, Kenosha, Wisconsin, public schools; M. D. Steer and James Maddox, Purdue University; Margaret Johnson, Topeka, Kansas, public schools; Robert Milisen, Indiana University.

on others to do things for him and to solve his problems, so that the child feels inadequate to solve his own problems successfully.[2]

TABLE I

POSITION IN THE FAMILY OF 522 STUTTERERS AS COMPARED TO THE NORMAL POPULATION*

Position	Stutt. (%)	Pop. Norm. (%)	Diff. / σ Diff.
Only	20.01	12.	4.48
Oldest	25.29	26.	.36
Youngest	27.01	26.	.50
Middle child of three	6.89		
Second child of four or more	6.90		
Third child of five or more	2.50		
Fourth child or six or more	2.11		
Next to youngest of four or more	6.70		
All other middle positions	2.49		
Total of all middle positions	27.59	36.	4.14

*Population norms taken from the White House Conference on Child Health and Protection, Section III-A; 7,738 children of junior high school age.

Table II shows that the mean number of years between stutterers and their nearest siblings is significantly greater (CR, 6.32) than the mean number of years between pairs of the non-stuttering siblings. Analysis on the basis of groups (oldest, youngest or middle) and on the basis of age differences between the children

TABLE II

MEAN NUMBER OF YEARS BETWEEN CHILDREN, FOR 522 STUTTERERS AS COMPARED TO THEIR NON-STUTTERING SIBLINGS

Mean No. of Years Between Child and the One Succeeding Him						Mean No. of Years Between Child and the One Preceding Him*					
	Stutterers		Non-Stutt.		Diff.		Stutterers		Non-Stutt.		Diff.
Group	N.†	Mean	N.	Mean	σ Diff.	Group	N.	Mean	N.	Mean	σ Diff.
Oldest	130	3.32	103	2.71	2.35	Youngest	140	4.05	82	3.07	2.93
Middle	143	3.63	275	2.68	4.30	Middle	144	2.97	296	2.58	1.89

*Considering each group as a whole the means were, for stutterers, 3.6 (for 557 age differences) and for non-stutterers, 2.7 (for 378 age differences). The critical ratio of this difference was 6.32.

†N. refers to the number of age differences calculated rather than the number of subjects involved.

and the sibling preceding them as contrasted to the one succeeding them, showed that for the middle children, who were stutterers, there was a greater number of years between them and the child succeeding them than the one preceding them. Of the 405 stutterers studied, who were not only children, 187 of these were separated, on one or both sides, from their siblings, by four or more years.

[2]For a full discussion of pampering and its effects, see Adler (1).

If a child is separated by several years from his siblings, we can be sure, other things being equal, that the mother has more time and attention to give that child than if the age difference was less. This would suggest, to the present writer, that, on the whole, the stutterers were pampered more in early childhood than their non-stuttering siblings.

The question may arise that if these influences are important, why do we find so many stutterers whose parents remember that the onset of stuttering was around 1.5 to 2 years of age? Johnson (8) and Steer (15) indicate that the speech of these supposed stutterers of preschool age is apparently not significantly different from the speech of other children their own age, when their parents or teachers first regarded them as stutterers. This would bring up the possibility that the parent who would pay such special attention to speech that is not essentially different from the speech of other children would also be the type of parent who would pamper the child excessively, be over-solicitous for him, over-anxious about him.

An analysis of the sex of the sibling nearest by age to the stutterer did not yield any highly significant results. The sex of the nearest sibling by age to the stutterer was found, as was the sex of the non-stuttering child of families of two children, in which one child was a stutterer. Assuming that the chances are equal of any child being either male or female, a tendency was found in the direction of the nearest sibling being of the opposite sex. The CR of the difference between our findings and chance expectation was 1.4 for our analysis of two child families, and 1.2 for our analysis of the nearest sibling by age.

On analyzing the sex differences among the stutterers, it was found that the ratio of males to females, in the only child group, was 3:1, as compared to 5:1 for all other groups combined. In terms of percentages, 26 per cent of the only children and 17 per cent of all the other groups combined were female. The CR of this difference was 1.94. If we can take this difference to be reliable, it would suggest that the answer to the baffling question of sex differences, in the frequency of stuttering, might lie to some degree in differences in early training.

Summary

1. Statistically significantly more only children and fewer middle children were found among stutterers than among a presumably non-stuttering population.

2. The mean number of years between the stutterers and their nearest siblings was statistically significantly larger than the mean number of years between the stutterers' non-stuttering siblings.

3. There was a tendency for the sibling nearest in age to the stutterer to be of the opposite sex.

4. For the stutterers, the ratio of males to females in the group of only children was 3:1 as contrasted to 5:1 for all other groups combined.

5. Analysis of the data suggests to the author that there seems to be a direct relation between the development of stuttering in a child and the degree to which the child is pampered, in the sense in which that word is used in this paper.

Conclusions

There can be little doubt that the factors investigated here, namely, position in the family, number of years between siblings, and the sex of the nearest siblings are clearly independent of hereditary influences. It was found, however, that these factors stand in measurable relation to the incidence of stuttering, and if we are to consider the large percentage of only children who stutter as compared to the percentage of only children in the total population, we may conclude that the role of training, in the etiology of stuttering, is an important one.

This study, however, does not show how or to what extent these environmental forces play a role in the etiology of stuttering. An analysis of the case histories of eight stutterers and the results of a tentative therapy which treats the stutterer as a case of personality maladjustment rather than as merely a speech defective, is now in the process of being written.

BIBLIOGRAPHY

(1) **Adler, A.** *Practice and Theory of Individual Psychology.* New York: Harcourt, Brace and Co., 1924.
(2) **Adler, A.** Position in family constellation influences life-style. *International J. of Ind. Psych.*, 3, 1937, 211–227.
(3) **Adler, A.** *Problems of neurosis.* New York: Cosmopolitan, 1930.
(4) **Brill, A. A.** *Psychoanalysis: its theories and its practical application.* (2nd rev. ed.) Philadelphia: Saunders, 1914.
(5) **Busemann, A.** Geschwisterschaft, Shultächtigkeit und Charakter. *Zsch. f. Kinderforsch.*, 34, 1928, 1–52.
(6) **Fenton, N.** The only child. *J. Genet. Psychol.*, 35, 1928, 546–556.
(7) **Hardy, M. C.** Aspects of home environment in relation to behavior at the elementary school age. *J. of Juv. Res.*, 21, 1937, 206–224.
(8) **Johnson, W., et al.** A study of the onset of stuttering. Unpublished.
(9) **Jones, H. E.** In *Handbook of Child Psychology,* (C. Murchison, editor). (2nd rev. ed.) Worcester: Clark University Press, 1933, Chap. 13.
(10) **Krout, M.** Patterns of adjustment in twenty-six ordinal types. Read before the American Sociological Society and Midwestern Psychological Association, 1938.
(11) **Levy, J. A.** A quantitative study of behavior problems in relation to family constellation. *Amer. J. Psychiat.*, 10, 1931, 637–654.
(12) **Louttit, C. M.** *Clinical Psychology.* New York: Harper & Brothers, 1936, 276–291.
(13) **Rosenow, C.** and **Whyte, A. H.** The ordinal position of problem children. *Amer. J. Orthopsychiat.*, 1, 1931, 430–434.
(14) **Shoobs, N.** Position in the family and school achievement. *International J. of Ind. Psychol.*, (in press).
(15) **Steer, M. D.** Symptomatologies of young stutterers. *J. of Speech Disorders,* 2, 1937, 3–13.
(16) **Thurstone, L. L.** and **Jenkins, R. L.** *Order of birth, parentage, and intelligence.* University of Chicago Press, 1931.
(17) **Ward, A.** A study of one hundred only children referred to a child guidance clinic. Smith Coll. Stud. Soc. Work, 1, 1930, 41–86.
(18) **White House Conference on Child Health and Protection, Sect. IIIA.** *The Adolescent in the Family.* New York: Appleton Century, 1931.

STUDIES IN THE PSYCHOLOGY OF STUTTERING: XII.

Emphatic Breathing of Auditors While Listening to Stuttering Speech*

STANLEY AINSWORTH
State University of Iowa

Introduction

This study was designed to measure the effect of stuttering speech on the breathing of auditors. The auditors were both stutterers and non-stutterers. The point in making such a study lies in the opportunity it offers to ascertain the characteristics of the breathing of non-stutterers under conditions which at least approach the conditions which obtain for the breathing of stutterers during speech. It is reasonable to assume that any changes thus produced in non-stutterers' breathing are not the *maximum* changes that could be produced if the stutterers' subjective state could be more fully approximated by the non-stutterers.

Previous studies of stutterers' breathing, while not highly related to the present investigation, have involved the development of certain issues and techniques which have been adapted to the present problem. The main studies to be mentioned in this connection are those of Downton (1), Fossler (2), Henrikson (3), L. E. Travis (4), and V. Travis (5).

Subjects and Apparatus

The subjects used were divided into the following groups:

1. Twelve normal speakers, six of whom listened to two stutterers and six of whom listened to one stutterer.
2. Four stutterers, each of whom listened to one other stutterer.
3. Four normal speakers, each of whom listened to one other normal speaker.

All of the subjects were males ranging in age from 12 to 26 years. In the first group, an effort was made to get subjects who had had only casual contacts with stutterers. The stutterers used were the most severe in the clinic. More than one was used because of convenience and in order to get a large number and variety of moments of stuttering. The auditors chosen had had no diseases that would reasonably be regarded as having affected their breathing for purposes

*This study was directed by Wendell Johnson. All pertinent data and materials are on file at the State University of Iowa.

of this study, and they had never stuttered. In group two, two of the stutterers had been in the clinic for at least one year. The other two were used shortly after they arrived. One of the subjects in group three had also been used in group one.

The apparatus used for recording the breathing curves was an ink-writing polygraph designed especially for this experiment.[1] The paper was drawn across the writing surface by a constant speed motor and was moved at the rate of 14 millimeters per sec. Four Marey type tambours and four Marey pneumographs were used. The pens, connected to the tambour levers, were fine tubes which syphoned the ink from a trough. Smooth white paper twelve inches wide was used. Six lines were recorded simultaneously: two breathing curves for the listener and two for the reader; a line used to indicate moments of stuttering by means of a signal marker; and a time line divided into seconds by means of a meter-long pendulum wired to another signal marker. Examples of the type of tracing may be found in Fossler (2) and Travis (4).

The material read by the stutterers and normal speakers was chosen at random from available magazines and was different for each record.

Procedure

The listener and reader were each stripped to the waist. They were seated facing each other, and the upper pneumograph was placed at the level of the nipples; the lower one was placed at the level of the umbilicus. The subject was cautioned to refrain from any movement while the experiment was in progress, and was instructed to watch the speaker while the reading was going on. For the first minute or two, the subject sat still and a record was made of his breathing while at rest. At a signal, the stutterer began to read. When a moment of stuttering occurred, an assistant depressed a key as long as the stuttering lasted. The assistant faced the stutterer so that he could see as well as hear the moments of stuttering. The reading continued for about five minutes. When the reading stopped, the subject sat still for approximately another minute and a record was again taken of his breathing while at rest. In eight cases, the listeners were unaware that they were to listen to a stutterer until the reading began. In all cases, the stutterer did no talking until the reading began.

Analysis of Data

I. Group One: Normal speakers listening to stuttering.

A. Coefficients of variation were determined for both inhalations and exhalations of rest and of listening breathing. The formula for obtaining the coefficient of variation is $C.\ V. = \frac{100\ S.\ D.}{Mean}$. Since

[1]A complete description of the apparatus used is in the appendix of the copy of this report on file in the State University of Iowa Library.

it takes both the mean and standard deviation into account, the coefficient is comparable from case to case. The time consumed by each inhalation and exhalation was measured to the nearest millimeter, or to within .07 of a sec. For rest breathing, 321 exhalations and 317 inhalations were measured. For listening breathing, 1159 exhalations and 1165 inhalations were measured.[2] The most sensitive record of each listener was measured, thoracic curves were measured, in eight cases, and in four cases, abdominal curves were measured. Any extremely abnormal breaths that were obviously due to something other than listening to stuttering were not measured. (Examples—sighs, abnormal curves due to movements, etc.)

B. The ratio of the variability of inhalations to the variability of exhalations for rest and listening breathing was established. The formula used was $\text{I. E. V.} = \frac{\text{C. V. Insp.}}{\text{C. V. Exp.}}$.

C. The subjects in group one were divided into two sections—four subjects who knew beforehand that they were to listen to stuttering, and eight subjects who did not know that they were to listen to stuttering.

D. An analysis was made to determine the degree to which specific moments of stuttering accompanying those specific exhalations which were of greater length than the mean duration, were directly related to the lengthening of exhalation in the listener. The per cent of exhalations that were above the mean was found. By chance, the same per cent of these exhalations that are accompanied by moments of stuttering would also be above the mean. If the obtained percentage were significantly larger than the chance percentage, it would indicate that the stuttering moments are directly related to the lengthening. To be significant, the difference must be three times the standard error of the difference.

E. An analysis was made to determine if moments of stuttering accompanying the exhalation were directly related to abnormally long or abnormally short exhalations in the listeners. An exhalation more than two standard deviations from the mean was considered abnormal. Since a certain per cent of all exhalations were accompanied by moments of stuttering, by chance this same per cent of abnormal exhalations would also be accompanied by stuttering moments. The difference between this chance per cent and the obtained per cent was determined. If this difference were three times the S. E. of the difference, it would indicate that these abnormal exhalations were directly related to the moments of stuttering accompanying them.

F. A comparison was made between the first fifteen listening breaths and the last fifteen in order to discover any decrease in

[2]A detailed account of the analysis is in the appendix of the copy of this report on file in the State University of Iowa Library.

variability after the listener had heard the stuttering for about five minutes. Means, standard deviations, and coefficients of variation were established as in part (A).

II. Group Two: Stutterers listening to stuttering.

A. Coefficients of variability were determined as in I (A). For listening breathing, 215 exhalations and 213 inhalations were measured. For rest breathing, 117 exhalations and 115 inhalations were measured.

B. I.E.V. ratios were determined as in I (B).

III. Group Three: Normal speakers listening to normal speaking.

A. Coefficients of variability were determined as in I (A). For listening breathing, 225 exhalations and 228 inhalations were measured. For rest breathing, 102 exhalations and 99 inhalations were measured.

B. I.E.V. ratios were determined as in I (B).

IV. General Analysis.

A. The coefficients of variability for each group were compared with the coefficients of the other two groups.

B. I.E.V. ratios were compared in the same way.

C. Abnormal curves that could not be included in the above computations were measured.

Results

1. Group One: Normal speakers listening to stuttering.

TABLE I

MEANS, STANDARD DEVIATIONS AND COEFFICIENTS OF VARIATION FOR EXPIRATION AND INSPIRATION, DURING LISTENING AND WHILE AT REST; TWELVE NON-STUTTERERS LISTENING TO STUTTERERS' SPEECH

	REST BREATHING			LISTENING		
	Mean	S. D.	C. V.	Mean	S. D.	C. V.
Expiration.....	2.20 sec.	0.37	17.1	2.11 sec.	0.46	21.3
Inspiration.....	1.41 sec.	0.21	14.6	1.28 sec.	0.21	15.9

Nine of the twelve subjects had greater variability of exhalations while listening than they did during rest breathing. Eight had greater variability of inhalations while listening than while not listening. Two of those whose variability of exhalations was less during listening had greater variability of inhalations during listening than in rest breathing.

A. The average I.E.V. ratio was .89 for rest breathing. The ratio for listening breathing was .77, indicating that the variability of exhalations increased more than did the variability of inhalations.

B. For the four subjects who knew they were to listen to stutterers the average coefficients of variability were as follows:

Exhalations while not listening	17.8
Exhalations while listening	18.6
Inhalations while not listening	18.4
Inhalations while listening	17.4

For the eight subjects who were entirely ignorant of the experiment beforehand, the average coefficients of variability were as follows:

Exhalations while not listening	16.7
Exhalations while listening	22.7
Inhalations while not listening	12.7
Inhalations while listening	15.1

The variability of the latter group was greater for exhalations while listening; and there is a greater increase in variability while listening for both inhalations and exhalations.

C. The average variability for the first fifteen exhalations was 21.5. The average for the last fifteen was 20.9—a decrease of 0.6. Although this decrease is small, nine of the twelve had an average decrease of 7.7. The other three cases had large increases due to one or two abnormal exhalations in the last fifteen breaths.

The average coefficient of variation for the first fifteen inhalations was 12.8; for the last fifteen inhalations, 16.0—an increase of 3.2. Six of the twelve subjects had decreases in variability.

D. Of the 842 exhalations accompanied by moments of stuttering, 45.8 per cent were found to be above the mean. Since 42.4 per cent would be above the mean by chance, the difference is 3.4 per cent. The S. E. of the difference was found to be 2.2 per cent, yielding a critical ratio of 1.5.

There are 93 chances in 100 that the true difference is in the same direction as the obtained difference.

E. Of the listeners' 57 abnormal exhalations, 41 or 72 per cent were accompanied by moments of stuttering. Since by chance 72.6 per cent of the abnormal exhalations would be accompanied by moments of stuttering, a negative difference of 0.6 per cent was obtained. Since the S. E. of the difference is 6.1 per cent, the difference is not significant.

II. Group Two: Stutterers listening to stutterers.

TABLE II

MEANS, STANDARD DEVIATIONS AND COEFFICIENTS OF VARIATION FOR EXPIRATION AND INSPIRATION, DURING LISTENING AND WHILE AT REST; FOUR STUTTERERS LISTENING TO STUTTERERS' SPEECH

	REST BREATHING			LISTENING		
	Mean	S. D.	C. V.	Mean	S. D.	C. V.
Expiration	2.18 sec.	.39	17.6	2.17 sec.	.42	18.6
Inspiration	1.53 sec.	.39	23.6	1.47 sec.	.31	19.3

The exhalations increased 1.0 in variability, and the inhalations decreased 4.3.

The I.E.V. ratio was 1.28 for rest breathing and .98 for listening.

III. Group Three: Non-stutterers listening to non-stutterers.

TABLE III

MEANS, STANDARD DEVIATIONS AND COEFFICIENTS OF VARIATION FOR EXPIRATION AND INSPIRATION, DURING LISTENING AND WHILE AT REST; FOUR NON-STUTTERERS LISTENING TO NON-STUTTERERS' SPEECH

	REST BREATHING			LISTENING		
	Mean	S. D.	C. V.	Mean	S. D.	C. V.
Expiration.....	2.71 sec.	.41	15.3	2.56 sec.	.37	14.5
Inspiration.....	1.73 sec.	.24	14.6	1.58 sec.	.25	14.7

There was a decrease in variability of exhalations of .8 while listening, and an increase of .1 in inhalations.

The I. E. V. ratio was .88 for rest breathing and 1.03 for listening breathing. This indicates that inhalations changed more in variability than exhalations, an effect opposite to that of Groups I and II.

IV. General Analysis.

A. In comparing the three groups as to variability we find the following to be true:

1. Exhalations of Groups One and Two presented increases in variability while listening to stuttering speech. However, the increase in Group Two was not so great, and there was a decrease in the variability of inhalations.

2. Group Three gave results different from those of Groups One or Two. There was a small decrease in variability of exhalations and only an insignificant increase in the variability of inhalations.

B. In comparing the three groups as to I.E.V. ratios, we find the following results:

1. Groups One and Two presented decreased ratios while listening to stuttering.

2. Group Three, however, showed a decided increase in this same ratio while listening to normal speech.

C. Four abnormalities that could be traced to no other condition than listening to stuttering were detected. Three of them were of such character that they could not be measured as either exhalations or inhalations. In these three cases, breathing was suspended for 6.16, 6.23, and 16.24 sec. at the beginning of the stuttering. All three of the normal-speaking listeners involved did not know beforehand that they were to listen to stutterers. The fourth abnormality was an inhalation lasting 5.67 sec. following one of these suspensions of

breathing. It was not included in the computations because it was found that doing so gave a decidedly distorted picture of the variability of that subject's inhalations.

Summary of Results

Three distinct methods were used in gathering data. Non-stutterers listened to stuttering speech. As a check and a means of relatively evaluating these results, stutterers listened to stuttering speech, and non-stutterers listened to the speech of normal speakers.

1. The exhalations and inhalations of non-stuttering listeners and the exhalations of the stuttering listeners were more variable while listening to stuttering speech.

2. This increased variability did not occur while normal speaking auditors were listening to normal speakers.

3. In normal speaking listeners, this variability tended to decrease as listening continued.

4. If the normal speaker knew before listening that the speaker was to be a stutterer, the increase in variability was not so great.

5. Moments of stuttering which accompanied auditors' exhalations were apparently related (93 chances in 100 that the difference between such exhalations and other exhalations was a true difference) to the lengthening of exhalation.

6. Moments of stuttering which accompanied the abnormal exhalations (more than two standard deviations from the mean) were not directly related to the abnormal exhalations that were recorded.

7. The records indicated suspended respiration in three subjects (non-stuttering listeners) for durations of 6.16, 6.23, 16.24 sec., respectively, immediately following the beginning of speech by the stutterer.

Conclusions

With reference to the problem as stated in the introduction, it would appear that the method used for this study was valid. The results show that some change takes place in the breathing of non-stutterers while listening to stuttering. This change is in the direction of increased variability with regard to duration of inspiration and expiration. Thus, it is in the direction of the nature of the change in breathing which occurs in the stutterer during speech as has been shown by several previous studies of stutterers' speech breathing (see references cited in the introduction). It is reasonable to assume that the increased variability in the non-stutterer's breathing, while listening to stuttering, was accompanied by, or was an expression of, a change in the listener's subjective state which is to be regarded as a change in the direction of, and an approximation of, the subjective state of the stutterer to whom the auditor was listening. This indication was empirically supported by the fact that non-stutterers' breathing did not become more variable

while they were listening to the speech of normal speakers. The increased variability, when it occurred, would appear, therefore, to be a function of the listener's subjective state as affected by the stuttering heard, rather than a function of the mere act of listening to speech.

It is to be concluded, therefore, that the findings indicate that increased variability in breathing is one accompaniment, or one expression of, the subjective state present during stuttering, and that this can hold true even when it is observed in an apparently normal speaker. Insofar as the stutterer's subjective state can be induced by the act of listening to stuttering speech, the breathing disturbances occasioned thereby are as great, if not greater, in the normal speaker as in the stutterer (when the stutterer serves as an auditor). This is indicated by the fact that the variability of non-stutterers' breathing, while listening to stuttering speech, was increased more than was the variability of stutterers' breathing, while listening to stuttering speech. It is, moreover, not to be assumed that the increased variability of the non-stutterers' breathing, as recorded, represents the maximum degree to which their breathing could be disturbed if the subjective state of the stutterer could be more fully duplicated in their experience.

BIBLIOGRAPHY

(1) **Downton, W.** The effect of attitude on the speech breathing of stutterers. M.A. thesis, University of Iowa, 1934.

(2) **Fossler, H. R.** Disturbances in breathing during stuttering. *Psych. Monog.*, 1930, 40, 1–31.

(3) **Henrikson, E. H.** A study of breathing and vocal disturbances of stutterers. *Arch. of Speech*, Studies in Speech Pathology, I, No. 2, 1936, 133–149.

(4) **Travis, L. E.** *Speech Pathology.* D. Appleton Co., New York, 1931.

(5) **Travis, V. E.** A study of the horizontal dysintegration of breathing during stuttering. *Arch. of Speech*, Studies in Speech Pathology, I, No. 3, 1936, 157–169.

ELONGATION OF THE CLEFT PALATE TO GAIN BETTER NASOPHARYNGEAL CLOSURE*

JAMES BARRETT BROWN, M.D., F.A.C.S.
St. Louis, Mo.

In the repair of cleft palates one goal should be to obtain the best possible function of the soft palate. This will require pliable tissue, sufficiently long to meet the posterior wall of the pharynx (Passavant's pad) in the sphincter-like action of this region that closes the opening between the nose and throat. Although operations may be well executed and the palates may appear normal after operation, it is extremely exceptional that perfect speech is obtained. This persistent speech defect is probably due to a leak of air into the nose which may occur if there is an opening left of only a millimeter.

In efforts to insure complete closure, methods have been described for gaining length in the palate and for carrying the posterior pharyngeal wall forward. These procedures have been completely summarized by Dorrance in "The Operative Story of Cleft Palate" and need only mentioning here.

Elongations have been done by freshening the edges of the posterior pillars, detaching them low down in the pharynx and then suturing them together in midline, thus extending the palate behind the uvula; this has effected good results in many cases, but tissue that is normally a tensor of the palate is changed around so that the levators are expected to pull it up in closure of the opening. This procedure puts a purse-string scar band right across the part of the palate that should be the softest and most movable, and, by contracting downward, may actually prevent normal elevation by the levators.

Rectangular flaps from the cheeks have been put across the palate after it has been dissected backward, but the procedure is somewhat formidable for the gain involved. Gillies and Fry have described a backward dissection of the palate with an apparatus to hold a skin graft on the raw surface, the resultant opening into the nose in the front part to be closed by an obturator.

Others have also sacrificed the closure in front in order to have the tissue available posteriorly and have then used obturators for the anterior opening or have brought in flaps from a distance. Some

---

*From the Department of Surgery, Washington University School of Medicine, and the Central Institute for the Deaf, St. Louis, Mo.

Reprinted in part from *Surgery, Gynecology* and *Obstetrics*, Vol. 63, p. 768 1936, and from the *American Journal of Orthodontics and Oral Surgery*, Vol. 24, p. 878, Sept., 1938.

think that in complete clefts an operation of the Dieffenbach principle may be executed to allow moving backward of the entire palate, but, if any great displacement is done, a hole persists in the front part of the palate.

After closure of the palate, several surgeons have directed their attention to carrying the posterior pharyngeal wall forward: by reefing it forward, by the use of a submucosal prosthesis, and by rectangular mucous flaps dissected free from the pharynx and attached to the soft palate. Any of these measures, especially the reefing forward of the posterior wall, which has been called pharyngoplasty by Wardill, can be added to the procedure described here, if the surgeon thinks extra function might be gained.

Palates that are cleft only through the soft tissue or on up, part way through the bone, may have actually less tissue for repair than those with complete clefts; and there are also patients with uncleft palates that are so abnormally short that speech is as bad as though the palate were cleft. Dorrance has called this "congenital insufficiency of the palate."

It is in this group that the most direct attempts have been made for actually lengthening the palate, by moving the whole of the soft tissues backward and still having the bony palate in front to separate the nose and the mouth. Dorrance's procedure which he calls the "push-back operation" is probably best known. The essentials are that a delayed flap of the entire covering of the bony palate is made except that the major palatine arteries are sacrificed. At a later operation the mucoperiosteum is again raised from the bone and the whole mass is "pushed back" so that the anterior edge of the flap may be anchored as far back as the posterior bony edge. The cleft is closed and the anterior denuded bone is left to heal over. This operation allows thickening of the tissues to take place, and this has been thought to be desirable by Dorrance to allow for freedom of manipulation in "pushing" the palate back.

Author's Technique for Lengthening the Palate

The principle of the procedure outlined here is that a direct flap of practically the entire palate is raised completely free from the bone and is immediately set back so that the anterior free edge is anchored all the way back at the posterior edge of the bone. The major palatine arteries are definitely preserved and left to supply the palatine flap. The palate is allowed to heal here, and the bony palate to cover completely with epithelium. The palate cleft itself is closed either at the same time or at a second operation. It is thought, by this immediate elongation of the palate tissue, that is, lifting the whole thing up and setting it back as far as possible at one operation, that excessive thickening can be avoided, and, therefore, that the best motility may be maintained. This point seems extremely important in consideration of the rapid movements required of the naso-pharyngeal closure in speech.

The details of the operation may be followed in the legends of the illustrations.

FIG. 1

FIG. 2

FIG. 1. Line of incision and beginning elevation of mucoperiosteum.

FIG. 2. Diagrammatic representation of palate completely detached from bone, both major palatine arteries intact, and preservation of a band of nasal mucosa to which the palate is attached with the first suture as shown.

FIG. 3

FIG. 4

FIG. 3. Detail diagram of deep separation of the soft tissues, exposure of the hamulus, freeing of the palate aponeurosis in this area behind the artery, section of the tensor tendon.

FIG. 4. Completion of first stage. The palate has been set back and anchored with horsehair sutures as shown. Several layers of iodoform and balsam of Peru gauze are placed smoothly over the exposed bone and will be retained without sutures for several days.

Complete healing of epithelium over the area denuded anteriorly occurs in twenty to thirty days, and is practically normal in appearance except that rugae are not present. At this stage the soft tissues may be somewhat humped up from side to side just behind the edge of the bone and the actual lengthening might be said to be the difference between what tissue is used in this humping up and the total distance the edge is set back. This distance may be as much as four centimeters and is the space between the anterior incision and the posterior edge of the bone. Improvement in speech is usually already noted even when the cleft is not completely closed.

The cleft in the palate is closed at the same operation if possible so that the entire palate is replaced posteriorly and closed in a one-stage procedure. However, in some children and in nearly all adults the bleeding is so profuse that two operations are advisable.

Results of Treatment and Qualifications of the Procedure

(1) No claim is made that perfect closure is to be routinely expected, but it is thought that results thus far have been very encouraging. Speech training is considered as important as in any other type of palate closure. No deaths have occurred from the operation.

(2) If there is interruption of the blood supply of the major arteries either when operation is done, or if a previous operative attempt has sacrificed them, it will perhaps be best to revert to a "delayed flap" type of operation, rather than to set the palate back at the first operation. Palates badly scarred from former operations cannot be expected to submit to manipulation as readily as unscarred ones.

(3) If there is a bony defect extending up the center of the palate, very careful separation of the flap should be done to preserve the nasal mucosa, otherwise a hole will be left anteriorly. The limits of the bony edge or the possibility of a long, narrow central cleft can be determined with a fine hypodermic needle if desired before operation.

(4) If the operation is done too early in life, any tooth buds outside the alveolus may be damaged, as has been emphasized by Logan and Kronfeld; therefore, the operation should not be done before the first or second year. Other operative procedures carried out, in this and foreign countries, too early in life may also result in damaged dentition and subsequent greater trouble with speech.

(5) A possible objection to this procedure is that the resulting raw nasal surface may contract and shorten the palate secondarily, but so far we have not noted this as being important enough to try to cover the raw surfaces with skin grafts. These palates usually appear excessively long on inspection, and the surface nearly always smooths out.

(6) Both the elongation and the palate closure may be completed in one operation, but the two separate stages are relatively easy ones for the patient, if thought best to separate them.

FIG. 5

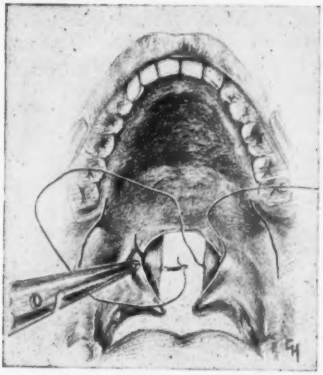


FIG. 6

FIG. 5. Beginning of second stage. New openings through the former lateral incisions may or may not be thought necessary. The edge is trimmed off from the tip of the uvula up into the palate substance about 2 mm.

FIG. 6. A deep stay suture is usually placed to engage a good bulk of tissue. This may be of catgut and tied on the nasal surface or it may be of nonabsorbable material and brought out into the mouth. Extra muscle and nasal mucosa sutures may be put in at this stage.

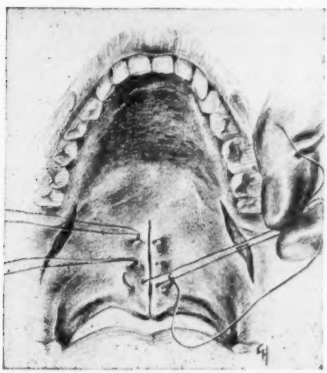


FIG. 7

FIG. 8

FIG. 7. Closure of the cleft with vertical mattress sutures of horsehair. The closure is carried entirely around the uvula and then up onto the nasal surface with fine catgut sutures.

FIG. 8. Completed and healed palate. Set back the distance of the original exposed bone shown in Fig. 4, minus the folding of the soft tissue and the contraction in healing. It is not possible to tell by looking at the palate whether or not it completely closes the nasopharynx, but, of course, the greater its length, the better is its chance of doing so, provided the levator muscles are functioning.

(7) This procedure seems to be adaptable to a fairly large percentage of the total cases, because its steps may be changed around, or combined in one operation, and because the first stage may be used to elongate clefts that have been closed a long time but still need more length, as evidenced by poor speech.

(8) It is also possible that palates with complete clefts that have been closed can be lengthened by preserving the nasal mucosa anteriorly to permit of spontaneous healing over the oral surface, the same as illustrated here over the bone.

(9) In a few patients, where there has been a marked lack of tissue and of bony process present, one elongation has been done and then, when healing was complete, further elongation was obtained by carefully dissecting the palate loose a second time and preserving the nasal mucosa to avoid an opening into the nose.

(10) A few examples of individual results are included here. All the older patients have expressed great appreciation and have felt definite improvement in speech. One western ranch hand, 25 years of age, undoubtedly the subject of much ridicule because of poor speech, stated that he was able for the first time really to get by with his work and to direct others at work following closure and elongation of the palate and speech-training.

One man of 30 years who had never had his palate closed, following an elongation, was able to teach Sunday School and to carry on his work with few people realizing that there was any palate defect at all.

Another adult who had not been operated on before was able to make her name understood for the first time in her life.

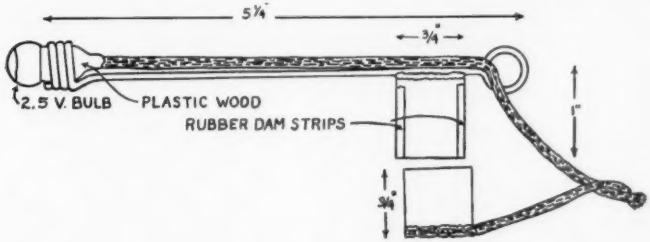
One girl of 12 was first seen many years after an attempt at palate closure, and showed total loss of all functioning soft palate—presumably the result of sloughing after the operation. The palate was set back as far as possible, and the remnants of the soft tissues were brought up from the sides and closed in midline as far back as possible. The result was then what amounted to a soft palate obturator made of her own tissue, but having no muscular elevation or other movement. With good effort and speech-training, this patient has developed remarkable speech, is able to do regular high school work, recite, and to sing quite nicely. She, incidentally, can put her tongue all the way back in her nasopharynx, which may have something to do with the good result.

(11) When this paper was presented at the Joint Session of the American Speech Correction Association and the National Association of Teachers of Speech at Cleveland, Ohio, on December 28th, 1938, electrical transcriptions of the speech of these patients were played (together with the recording of speech in one person with complete absence of the tongue following removal for carcinoma). These records were made in the Sound Laboratory of the Central Institute for the Deaf, Saint Louis, and the author wishes to express his appreciation to Miss Mildred McGinnis, Dr. Max Goldstein and Mr. C. E. Harrison for their help and interest in the work, and to the other teachers of speech who have worked with the patients.

A SIMPLE ORAL EXAMINATION LIGHT

JACK C. COTTON, Ph.D.,
Director, Speech Clinical Laboratories,
Western Washington College of Education,
Bellingham, Washington

In setting up our speech laboratories here at Bellingham[1] one problem to be solved was that of getting adequate light into the mouth of each student so that movements of the tongue and velum can be clearly seen and studied during the various articulatory processes. Ordinary room light for mirror examinations of the oral cavity is highly unsatisfactory. The rather expensive diagnostolites were out of the question for us since twenty-four students work simultaneously during a laboratory hour. The idea of using flashlights was discarded because of expense and ineffectiveness of flashlights in general for this purpose. All but the smallest flashlights interfere with the view of the mouth, and in these small flashlights battery replacement becomes a constant nuisance.



The simple lights finally designed and constructed for group laboratory work involving self-examination are equally well adapted for speech clinical examination purposes. Their construction is simple and perhaps of enough general interest to warrant publication of the accompanying drawing and construction details. We have about forty such lights, built with very little expense, all functioning very satisfactorily.

The handle and bulb support was made from No. 12 gauge galvanized iron wire. Brass wire could be used if desired, since this can be nickel-plated and given a more finished appearance. The wire was bent into a single loop at one end, around a ⅜ inch rod. This same rod, roughly reduced by about 1/32 inch in diameter by filing,

[1]Cotton, Jack C. A Laboratory Program in Speech Education, *Western Speech*, 3, 1939. 1–5.

was used to twist the three loops which serve as a socket for a two-cell flashlight bulb.

We used ordinary twisted light cord for wiring these lights. One wire was scraped clear of insulation for about 1/16 inch, soldering paste applied and this soldered to the center contact of the bulb. No solder was needed beyond that normally found on this contact. The wire was brought back parallel to the wire handle and taped to the handle just in front of the handle ring.

Bulbs were soldered in this way to avoid the extra trouble and bulk involved in placing and insulating a contact for the center of the bulb base. Furthermore, this procedure eliminates loss of bulbs taken for student flashlights. Replacement of bulbs involves more than simply screwing in a new bulb, of course, but when four or five burned out bulbs are replaced at once the time required is not excessive.

A small pellet of plastic wood was worked over the soldered joint and the base of the bulb, allowed to dry, then given a coat of white enamel. This prevented the slight shock possible without this protection. At the same time the bulb was given a coat of enamel around the sides, only the end being left free. This masks those rays which otherwise glare directly from the filament into the observer's eyes. After applying these touches of enamel the lights were allowed to dry clamped vertically, bulbs up, to prevent the enamel running down onto the clear part of the bulb or forming soft blobs on the sides.

At first, we made no provision for switching off the light other than by pulling the wires out of the current-supply jacks. Bulbs were allowed to remain on when not being used, so that bulb replacement became a real problem. To preserve their lives and our time the simple finger pressure switch shown was added to each light. A 1 by 3/4 inch piece of No. 30 gauge sheet brass was soldered near the end of the handle. The other light wire was soldered to a 3/4 inch square piece of the same material. These two parts are shown separated in the cut. These pieces, separated by thin strips of rubber dam at the edges, were bound together with adhesive tape. One piece of tape was wound around in a horizontal direction (with respect to the drawing) another encircling the switch and wire handle above. The top piece of brass was bent inwards slightly wherever necessary to make the switch sensitive to slight finger pressures.

These lights can be used with two dry-cells or any 2½ to 3 volt current supply. We use a single 2½ volt step-down transformer to supply current for twenty-four of these lights connected in parallel. Others are used with batteries.

After trying a variety of sterilizing solutions we have settled on a 1/5000 solution of bichloride of mercury. The lights are washed in running water after use, then allowed to remain in the sterilizing solution for an hour before a final washing. This solution, unlike several others tried, does not affect the enamel on the lights.

TREATMENT OF STUTTERING

GEORGE A. KOPP
Director of Speech Clinic,
Wayne University

In a discussion of any problem in speech correction it is important that we remember that we are partners in this business of speech education. It is unfortunate if some of us are selfish enough to believe that our methods belong to us, that they must be used exclusively by us, in our schools, for our pupils only, and for our individual benefit and professional advancement. We have all profited by the rapid growth and expansion in speech correction during the past decade. Because of our effective promotional activities, educators throughout the country have become speech correction-conscious. We have constantly centered our attention upon correcting, re-educating, and rehabilitating the speech defective. Our cry has been for larger and larger speech correction departments. So effectively have we sold our program that I hesitate to call your attention to the fact that our educational foundation is somewhat unsound. Our growth and expansion to a great degree is directly due to the inefficiency of the regular classroom teacher. Our case load could easily be cut in half if the elementary teachers were trained to assume the responsibility that is justly theirs. It seems to me that this is an opportune time to change our emphasis from correction to correct education, from reeducation to education.

This would necessitate a shift of emphasis from the large enrollment in our classes to the records showing the number of speech defectives rehabilitated. Likewise because of our excellent promotional efforts our methods of treating stuttering will sooner or later be scrutinized by the informed administrators.

We all know that the stutterer has been our professional guinea pig for many years. Through these years he has been our chief source of publicity. Sometimes I feel as though he has given the profession more than we have given him. Yet, when I recall the combined efforts of the members of our associations to find a solution to the stuttering problem, I am deeply impressed by the amount of time, energy, and money that has been expended in behalf of the stutterer. The results of our work may be questioned but we cannot question the fact that because of these studies many of our most influential partners have become more deeply entrenched in their own individual theories and practices. The methods for treatment of stuttering still remain as varied as the theories concerning the etiology of the disorder. This explains why the methods used in every school system are

those of the universities in which the teachers were trained. Too frequently this training has been narrow in scope and limited to a single point of view. In some institutions the training includes little or no methodology. We have become professional jitterbugs in respect to this impediment. We do not know what to believe nor what to disbelieve. One year we are impressed with one theory, the next year with another theory, and collectively we present to the educational world a disconnected and disorganized front. We seem to have definitely agreed to disagree.

I plead guilty to contributing to the confusion, for, as you know, I have questioned every theory of stuttering that has ever been advanced. I have done this because I was convinced that no one theory or method of treating stuttering is sufficient to correct the speech of all stutterers. Nor is any one method adequate to correct the speech of the majority of stutterers. In ten years of working with stutterers I have never been able to completely cure a single adult stutterer, using one or several approaches. It is true that I have helped to improve the speech of a number of stutterers, both young and old. But I would hesitate to assume much credit for what happened to any of them. Dr. West had an apt way of distributing the honors, if any, for what we did with stutterers at Wisconsin. He would say, "Oh sure, God, nature, the patient, Kopp, and myself have helped a few stutterers." I cannot help but believe that these other factors are vitally important, regardless of the method used. It may sound absurd to you but I can honestly say that I have never seen an adult that any of you have completely cured of stuttering. You may have cured thousands of adult stutterers but I am merely saying that I have never happened to see one that I would say was completely cured.

Then again I have questioned all theories because there has been a failure to evaluate the detrimental effects of some of the therapies that are in use and there has also been a striking absence of impartial follow-up studies to show definitely what percentage of the so-called cured stutterers retain their speech proficiency. Also, the therapies as they are practiced are seldom limited to a single approach such as the physiological, psychological, sociological, neurological, psychoanalytical, pedagogical, or any other one point of view. This is as it should be, but why claim credit for one method when several are being used? The fact that in practice we recognize the merit of other methods is important to us today. If we could only agree that each point of view has some value for certain stutterers, that each alone is too restricted and limited to be universally accepted as the best method to use for all stutterers, we could establish a basis upon which we could formulate a system of therapy that would be acceptable to all.

To do this we would need to agree that there are many possible causes of stuttering. If we accept the probability of this being true, we would necessarily have to broaden our concept of methodology to include the entire personality. The method or methods we would

then use with a given stutterer would depend upon a diagnosis of his personality.

Doctors Haggard and Fry[1] of Yale University, in their excellent book titled "The Anatomy of Personality," have dissected the personality for us. They do not mention stuttering or the stutterer but their analysis of the personality can be of great value to us. These doctors maintain that the component parts of any personality include:

a. The physique.
b. The impulse or energy drive.
c. The intelligence.
d. The temperament.
e. The ego.

If we consider the stutterer from these five points of view, we are thinking of him as an integrated individual. With this concept we can appreciate the possibility of more than one predisposing cause of the changes in metabolism that may result in the tonic or clonic oral myospasms that we call stuttering. The maturation level of the individual stutterer in respect to these various personality factors becomes most significant. Such an approach provides some insight to the internal environment of the stutterer, and offers a basis upon which all accepted practices in the treatment of stutterers can be orientated.

Let us consider each of these five factors of the personality and illustrate how the various schools of thought concerning stuttering may be applied for some stutterers but not for all.

First, look at the physical development and state of health of the stutterer. We are all concerned with his general health and hygiene. We stress relaxation, resting, eating, and sleeping habits. We use games of various kinds. We note any endocrine disturbance, abnormal sex manifestations, and check the general metabolism. We can all give examples of stutterers whose speech has responded to improvement of health. Especially is this true of children. Concerning the sex ratio of stutterers, I have reached the conclusion that if we did not have this ratio we would indeed have something to explain. Research has proved that there is a difference between the sexes in regard to seeing, hearing, and heart rhythms, so we should expect this difference to be manifested in the speech of the stutterer. I am ready to accept this as a fact and I am thankful that it isn't otherwise.

The impulse or energy drive of the stutterer, like that of the normal speaker, is centered in self-protection, self-development, and all self-interests. The strength of the impulse is determined by the physical vigor. And we know that the impulse life can be sublimated but not suppressed. We frequently see stutterers whose

[1]Clements C. Fry, M.D., and Howard W. Haggard, M.D., "The Anatomy of Personality." Harper and Brothers, 1936.

impulse life or energy is misdirected. The establishment of sociably acceptable goals and outlets is of great importance in treating such stutterers. I am sure that the sociological therapy has long been practiced with commendable results with certain types of stutterers. I have used it myself with some degree of success.

The mental state of the stutterer has long been investigated and we need but mention cerebral dominance, temporary auditory amnesia, faulty visualization, brain waves, configurations, and others to understand that this phase of the stutterer's personality has not been neglected in research or in therapy. Here again I can truthfully say that I have shifted the motor lead of a few stutterers with definite improvement in speech and general health. But there are only certain stutterers with whom we can use this therapy.

Next examine the temperament of the stutterer. His tempo frequently manifests great inner tension. He is excitable, irritable, and often over-anxious. His mood, like that of everyone, reflects the essential harmony or disharmony of the fundamental processes. The sensations which establish the mood arise from the activities of the cells of which the body is constituted. This had led a few of us to believe that in some stutterers the vital activities of the body are altered because this is the only known way of altering moods. The mental and emotional balance, or preferably the failure of the maturation of this balance, has long been a theory for the cause of stuttering. No doubt many of you here today have explained stuttering as an absence of cortical control over impulses from the thalamic region. And I am ready to agree that this is a good explanation of the cause of stuttering for some stutterers.

The fifth and last factor of the personality is the ego. In ego development we have a testing of individual strength against environment. We have observed that a few stutterers seem to find the environment too strong for them, or perhaps I should say their ego is too weak. The weak ego is characterized by timidity, self-consciousness, and sensitivity. These are good descriptive terms that apply to quite a few stutterers. So once again we find that this phase of the personality has been utilized in the treatment of stuttering. Surely a weak ego can be encouraged. Give the stutterer confidence in himself, build up his self-esteem, and note the change in his speech. We have all recognized this fact and we have used it, but once more let me repeat that all stutterers should not be so treated. In some the ego is too strong and may need to be curbed or discounted.

It is obvious from this brief discussion that every acceptable method for treatment of stuttering deals with one or more of these five personality factors. Our point of view has been too frequently limited to one or two of these factors. To be thorough we must deal with the whole individual. We are partners in this business and it seems to me that until research proves otherwise we would do well to use that method or methods that will most effectively apply to the various factors of the personality with which we are

dealing. We must make our examination of the stutterer from these different points of view, keeping constantly in mind the age and maturation level of the patient. These factors are especially important in working with stutterers in classes. If one and only one phase of the personality is considered it is reasonable to assume that the majority of pupils in the class will derive little if any benefit from the instruction. It is almost imperative that group instruction be supplemented with private and individual help.

In conclusion, I reiterate my conviction that much of our expansion in speech correction during the past decade has been due to the inefficiency of the regular elementary teacher who has refused to accept her responsibility to help the children to correct baby talk, lisping, and other common sound substitutions and sound omissions. Speech correction classes are filled with pupils who have these simple language difficulties; consequently, we have less time to give to the more serious speech defective. Our task is to see that the elementary teacher of the future receives the necessary training that will enable her to assume the responsibility that is hers. Our concern should be education rather than reeducation or correction. In working with speech defectives it seems to me that we should take professional pride in the number that we rehabilitate rather than the number we have registered in our classes. Our methods for treatment of stuttering have been incomplete and in many instances inadequate mainly because we have been considering only a part of the whole personality. If we work with the entire personality we could combine our methods, thus utilizing the best that each school of thought has to offer toward the common goal of improving the speech of the stutterer.

THE TREATMENT OF STUTTERING

Contribution of WENDELL JOHNSON to the Panel Discussion at the Cleveland Meeting

The longer I work with stuttering the more tolerant I become of any one who has any therapeutic ideas at all concerning it. Dr. Kopp pointed out that the stutterer should not take himself so seriously. Being both a stutterer and a specialist on the subject, I am sure not only that the stutterer should not take himself seriously, but also that the specialist should not take himself seriously either. There is no such thing as *the* method for treating stuttering, and the fact that so many different methods are more or less successful is of far more than incidental interest. We need not, however, carry our tolerance to the point of uncritical acceptance of every therapeutical approach with the same degree of enthusiasm.

For the purpose of this discussion, I should like to focus our attention upon one general point of view which I might introduce by referring to the remarks made by Mrs. Stoddard. To make a very blunt confession, I used to feel that Mrs. Stoddard was "talking through her hat," but I want to make it clear today that I think she has some very sound ideas. In her remarks here today, it seemed to me that one thing stood out as being of particular significance, namely, that she approaches a stutterer therapeutically with the assumption that it is possible for him to speak in an acceptable or normal manner. In championing this assumption, she brings sharply to our attention the issue upon which we tend to divide definitely and significantly. It is likely that the members of the Association could be divided into two groups, those who operate on the assumption that there are stutterers—the assumption, that is, that stutterers are different in some organismic way, aside from their speech, from non-stutterers and that this difference is present actually or potentially at the time of birth—and those who operate on the radically different assumption that either stutterers as a group are not different from non-stutterers as stated above, or that if they are different, the difference is not great enough to make a difference with respect to the stutterer's prognosis. I do not feel the necessity of drawing too sharp a line between these two groups and of contending that one must always adhere to one or the other of these basic assumptions, but I should like definitely to state my preference, based upon results to date from research and clinical experience, for the assumption either that stutterers as a group are not different in any basic sense from non-stutterers aside from their speech behavior and evaluations of that behavior, etc., or that if they are different, nevertheless, they are alterable to the extent that one is justified in operating clinically on the assumption that they can become non-stutterers.

You will remember that Mrs. Stoddard made quite a point of the desirability of trying to convince the stutterer of his ability to speak in an acceptable manner. I want to endorse her insistence on this point. My reasoning here can be made more clear perhaps if I point out that, while in the past we have been zealous in our efforts to check the validity of theories of stuttering advanced by various experts, we have almost entirely forgotten to investigate the theories of stuttering advanced by the stutterers themselves. I suspect that the theory of stuttering or of speech held by a given stutterer—or a normal speaker—might well determine, to some degree at least, the way in which he speaks. If we examine a stutterer from this point of view, we are immediately struck by the elaborateness of the set of assumptions which he holds regarding speech and stuttering, and it becomes quite plain that, insofar as he is acting on these assumptions, he can hardly speak in other than a hesitant and labored manner. We are also struck by the great probability that a large number of his assumptions are invalid and it is difficult not to suspect that the elimination or drastic modification of his assumptions might well result in more fluent speech. On the basis of the preliminary assumption that he is somehow abnormal, pathological, unable to function normally, the stutterer may and does erect any number of further assumptions to the effect that some sounds are more difficult to negotiate than others, that certain words are especially hard to say, that he will stutter relatively more or less to certain listeners or in certain situations or under the influence of such conditions as loss of sleep, changes in the weather, changes in going from one environment to another, etc. I want to suggest the extremely simple point that an individual, quite by definition, will remain a stutterer so long as he holds to such assumptions.

In this sense, the stutterer reminds me of a landlady I once had. Upon taking over her house I noticed a beautiful clock on the mantel and asked her if it was in working order. She replied that there was something the matter with the old clock and that it had not run for over eight years. She did not seem to know exactly what was the matter with it, but she was thoroughly convinced that it was out of order. Out of sheer curiosity I went to the clock and proceeded to wind it. It started to run immediately and continued to work very adequately during the entire time that I lived in the house. I am inclined to suspect that most stutterers and many speech pathologists formulate and act upon their assumptions regarding stuttering in a manner not unlike that of this curiously uninvestigative landlady.

After all, the problem in the treatment of stuttering is that of altering certain aspects of the stutterers' behavior. It seems to me desirable to begin always with the assumption that they are alterable. It also seems to me clear that a behavior, insofar as it is modifiable, can be altered either directly, or indirectly through changes in the assumptions which underly the behavior. Moreover, it is true not only that a change in assumptions results in a change in overt behavior, but also that a change in overt behavior results in alteration

of corresponding assumptions. That is, you can quite literally talk a stutterer into the conviction that he is able to speak more fluently than he does—I assume that we are all so familiar with this fact that it would be needless to labor it—or we can, after the manner of Mrs. Stoddard, tell the stutterer to behave in some way different from his customary behavior. So far as he succeeds in following directions of this kind and finds himself exhibiting new types of behavior, he will in time express a corresponding change in his assumptions regarding his possibilities for action.

One need not be too concerned over the question of how one is to change overt behavior. At a recent psychological meeting, Dr. Donald Lindsley presented the case of a man who had somehow learned voluntarily to raise the hairs on his arm. When asked how he did this, the man replied, "How? I don't know. How do you raise your arm?" The point is that in such a question the word "how" actually means little more than "what." If you were to ask Hank Greenberg how he hits a home run, all he could possibly tell you is what he does in taking up a baseball bat, going to the plate, pulling it back, swinging it forward, and hitting the ball. When you have his description of what he does, you know all that you are going to find out about how he does it. There is no mystical "how" about talking without stuttering. It is only a matter of the stutterer knowing precisely what to do—and preferably no more precisely than a normal speaker knows—and then attempting to do it. It would be my suggestion that if these attempts are not successful, it would be a sensible procedure to alter any assumptions the stutterer has which are operating to keep him from altering his behavior in the desired direction. Assumptions are seen in their overt form as statements made by the individual and it is, after all, these statements made by the individual which have to be recorded, examined, subjected to a procedure of validation and altered in the directions indicated by this procedure.

It seems to me that practically all of us have been following for years the general approach that I have tried to describe. I feel it important, however, that we recognize thoroughly all the time that, regardless of the specific clinical techniques we may use, our aim is either to alter the so-called stuttering behavior by means of directed instructions, or to alter it indirectly by altering the stutterer's assumptions regarding it, and above all I should like to make the obvious but highly important observation that so long as an individual retains and operates on assumptions regarding stuttering and speech, which are characteristic of stutterers, he will not become the sort of individual we refer to as a normal speaker.

CAN A CHILD BE TAUGHT TO TALK?

SARA STINCHFIELD HAWK,
University of Southern California and Hill-Young School

Speech development with its various problems, delays, physical, mental and emotional aspects, has rather recently begun to receive the serious attention of workers in the psychology and pathology of speech Investigations of such workers as Dorothy McCarthy, Blanton, Travis and Orton, Bluemel, Goldstein, West, Carr and Kennedy, point to the conclusion that the beginning of verbalization is a very complicated process in which the gradual acquisition of speech skill depends upon a combination of visual and auditory external stimuli and a linking of these with the kinaesthetic impressions and correct muscular movements necessary for reproducing sounds. This involves the building up of motor patterns, and we know that the finer accessory muscles cannot function in their fullest capacity until the nervous system has reached certain stages of development. It is true, however, that from around the fourth month the baby is babbling, prattling and accidentally making consonants and vowel sounds. It is true in speech as in other motor activities that a great deal more depends upon the way in which speech begins, than we have yet realized. False perceptions are as liable to be formed in speech as in any other type of reaction, and if a child builds up a faulty auditory or visual impression he tends to acquire the faulty habit at the onset. Once it is fixed, it takes far longer to eradicate it, than to train the infant into right habits at the outset. It is here that the speech specialist must begin to bring his work up-to-date, if he is to keep pace with similar important phases of research and training among medical and psychological groups. We still have many moss-covered, obsolete concepts in the field of speech in both the artistic and corrective field. The Italian maestro who recently caused a sensation by saying that he insisted that his students know the anatomy of speech organs, was no more recent in his concepts than Leonardo da Vinci, and yet many since da Vinci have failed to follow his example, unfortunately, in the field of anatomy and physiology.

After the larger fundamental muscles have perfected their co-ordinations, and are functioning as nature intended, about the end of the first year, the finer accessory muscles are ready for training. At the medical center in New York McGraw has found that about the end of the ninth month infants placed in water will easily acquire swimming movements, even though the head cannot be held up as at a later period. If they wait until after the 14th month, the child reacts much like a panic-stricken adult, and acquires these movements far

less easily. Also, in experiments with roller skating at the hospital they have found that the child acquires ease and skill more readily between the 14th and 18th month, than after the 18th to 24th month. The conclusion is that many habits are in the easy, malleable stage at an earlier period than we have been accustomed to believe, and that during this period of acquisition it is as easy to form wrong impressions and learn inaccurate habits as it is to learn correct ones. It is important that the child receive the right impressions and hear the correct models, if he is to acquire the desirable forms of speech and save much pain, humiliation and labor in later childhood or adulthood.

Seeing the movements as does the deaf child in learning to read the lips, or hearing the sound as does the blind child who cannot see movements, is not always able to create a strong enough impression in the child's perceptual centers, so as to enable him to reproduce in a motor way what he has gained from an external sensory stimulus. He must make mistakes, practise many times and train the muscles in order to reproduce sounds accurately in the serial order used in words and using the correct rhythmic patterns for sentences.

We are discovering in working with pre-school children in Los Angeles that the earlier the speech defects are discovered, the shorter the period of speech "incubation" necessary. There is almost a geometric increase, in the amount of training required in later life for speech-correction, according to the age of the child when the correction begins.

We are discovering every day that some children have become permanently retarded, very likely, due to early deprivation of speech training. Without speech the child is awkward, ill-at-ease, and if intelligent, he defends himself by turning against normal speech stimuli and shutting himself off from aggressive adults in a little world of his own. Being unable to mingle with the group on terms of equality, he often appears mentally retarded, and may even become so by deprivation—he is awkward in motor activities, and a social "outcast," from the standpoint of other children. The nearer he is to school age, and the more intelligent, the more he suffers and tends to become a behavior problem. Consequently we are emphasizing the importance of early recognition of speech defects in young children, before they have become set into speech patterns, or the child has turned against speech stimuli, visual, auditory and motor.

During a period of three or four years, I have been bringing to the various conventions the results of our research and study on the use of a new method of speech training, especially applicable to young children. This has been a long neglected field. While a good deal is known about the psychology of childhood, we have done little about speech pathology during the earlier years. The fundamental value in the material which I wish to present lies in the explanation of a learning process which is brought about through the definite direction of speech muscles by a trained adult. The learning is based upon kinaesthetic and motor learning.

The importance of this work was brought to my attention when I first went to California, and I subsequently made many visits to the Hill-Young School of Speech in Los Angeles, and later undertook research which has been elsewhere described, as we made studies of the physical status, intelligence, hearing and speech reactions of about 100 pre-school children in three different nursery school groups. The results obtained by application of Mrs. Young's therapy were felt to be outstanding, and it was a matter of considerable surprise to me to see children change from maladjusted, unsocial and unco-operative little folks, to normal and even to superior types, as they gradually acquired that most important outlet, symbolic speech. Further study and observation has convinced not only myself but many other observers that its widespread usage for the prevention and correction of speech disorders should be brought about by making the training available to large numbers of children everywhere. So outstanding has been the work that during the past year the Rockefeller Foundation has given a two-year grant to the school for the purpose of teaching the therapy and training teachers in its use. It is a therapy by means of which a child may actually be taught to talk.

Up to the time that the use of motor and kinaesthetic cues were brought into speech training, a child could *not* be "taught to talk." He was *aided* to gain speech patterns if he could make application of visual and auditory stimuli. Whether he succeeded or not in learning the words, depended upon his ability to move his mouth, tongue and throat muscles in a certain way in order to say the given word. There is no necessary connection between the ability to hear sounds in the sensory and perceptual areas for audition, and to make the correct motor movements necessary for reproducing the pattern heard. It may require definite direction, to enable the child to gain the correct muscular sequences of movement used in verbalizing. This means that there is a learning process going on, and that connections and associations have to be formed, before the child can voluntarily reproduce the sound impressions that have been stored in the brain, and which have been seen and heard. Some children are strongly audito-visual, or visuo-auditory, others are as surely audito-motor, visuo-motor, or moto-kinaesthetic in learning.

Now, we have formerly left the infant to acquire the correct muscle movements for himself, however complicated the pattern or sequence of movements involved. We have assumed that he was acquiring speech normally, because he could babble mama, papa or da-da. And if perchance he did not make even these sounds, we have accepted the time-worn admonition, "Let him alone. He'll outgrow it." The trial-and-error method thus employed might not be disastrous to the child, if each incorrect pattern could be erased after the unsuccessful experiment, but unfortunately it too soon becomes impressed upon the nervous system of the child, and the longer the wrong habits continue, the longer it will take to eradicate them. They are like "tics," grimaces and mannerisms of stutterers and extremely nervous persons. They may never wholly disappear.

Each bit of learning and each definite pattern by which the child learns to respond, when he says "det" for *get*, not only leaves a tendency toward repetition of the wrong muscular movements, but offers a fertile soil for conflict with the correct movement when it is established. There are certain factors involved in saying a word correctly, which have been much emphasized in the recent manual describing our experimental work at the Hill-Young School of Speech where several research workers are experimenting with methods and approaches to the speech difficulties and delays of childhood. In describing our *Children with Delayed and Defective Speech*[1] we have stressed especially the following, after careful research and actual work with young children which comes not merely from research itself, but from years of careful training and experimentation on the the part of Edna Hill Young, before her results were ready for publication:

1. That there is a definite *place* of beginning, for each sound with the musculature of the speech mechanism, and only that movement at that place will produce the desired sound. For instance, every word beginning with the sound of the letter "f" must be initiated by a movement of the lower lip. (Not as it is incorrectly described in some textbooks, as a bringing of the upper teeth down over the lower lip! Try this in your mirror, if you wish to test its absurdity.)

2. The second factor is a knowledge of how to move the muscles in order to produce the current sound for the beginning of a word. There are many things which may be done by the same muscles and correlated groups of muscles. How do we make the correct forms? At the outset there is no established tendency to move in any given direction. The infant may easily fail not only on the *place* of movement, but also upon the *form* of movement.

3. Third, the direction of movement can be made up, down, forward or back or away from the mid-line.

4. The 4th factor is the timing of the movements. This depends upon the ability of the child to move the correct parts through the correct movements in normal tempo—a thing for which he has certainly not been prepared by nature, except through a slow learning process, because he must give attention, concentration and effort to making the series of movements in the right order. Reversals of sounds and syllables are common in all children learning to talk, but their persistence is the thing to be avoided. An infant may fail in all three, that is in placing the movement, or in the correct beginnings, (2) in the form or direction of movement, and (3) in the timing of the movements in speech.

5. Pressure. You exert more pressure in producing a voiced than a voiceless sound, in order that the child may sense the difference between the vibrated and unvibrated sounds, and that in

[1]Stanford University Press, Stanford University, California, 1938. Stinchfield-Young. *Children With Delayed and Defective Speech.*

setting the pattern the trainer tries to give the clue by the degree of pressure exerted.

As we have already stated, the introduction of moto-kinaesthetic as a learning factor has changed the answer to the question "Can a Child Be Taught to Talk?" Under the direction of a trained adult, the *place* for beginning the movement may be indicated and sensed by the child through the kinaesthetic. The trainer gives the auditory pattern and necessary visual stimulus as he gives the beginning of movement, force of movement, direction, timing and pressure cues. As the trainer gives the auditory pattern, indicates where to begin by a sensory process, he also moves the part correctly at the same time, co-ordinating the auditory sensory and the moto-kinaesthetic impressions using the kinaesthetic as the cementing force which binds the two together inseparably in the child's mind. By this process of association the word is made easy and delightful for the child.

The change of direction is important. For instance the stutterer initiates the sound correctly, but he may remain quiet indefinitely, failing to release in normal tempo. So important do we regard the beginnings of stuttering that already Mr. Eugene Hahn, of the University of Southern California, is furthering the research program by concentrating on camera studies of children who stutter, to ascertain the seriousness of conflicting muscular patterns in the emission of sound sequences and in order to apply the motor-kinaesthetic techniques as a therapy in selected cases.

The infant learns only one word at a time in his progress. But bit by bit, he acquires parts of the whole complicated process of talking spontaneously. The recording mechanism of the child's own brain must be filled with many, many impressions before he can reproduce easily and fluently that which he has been long in acquiring. Witness an adult's struggle with a new language.

Longer sequences of two and three word sentences are further steps in the gaining of muscular control in the expression of ideas. By the trainer's direction of movements of the first words, other words may be gained without the waste of a preliminary trial and error period. We believe that the finest work might well be undertaken with all children at the 12th to the 15th month, in which we may assist the child in locating the place of movement, starting with the correct muscle sequences or forms for each word, and training the child in the timing, the whole pattern being set at one time. Immediate responses should not be expected or required.

The training must be in the casual nursery-school approach, free from tension or excitement, free from over-domination, and may sometimes need to be given as play, at the outset, while with other children a firm discipline needs to be maintained. Several training periods a day are far better than one protracted period. We have observed in watching Mrs. Young work in spontaneous practise of the play type with the child between training periods. "Foot ball" and "cup" are easy beginning words, which may be directed into the

child's vocabulary. These give him a variety of movements, and as you know, the child does not follow the phonetic alphabet in the order of acquiring his speech sounds. He does seem to acquire certain labials and tip of tongue sounds such as p, b, m, t, d and n more easily than many other consonants, as these movements are early used in the food-getting process. No attempt is made at first to have the child say the word. We present the object, talk about it, allow the child to play with it and then we gently name it several times, carefully and accurately, but not with undue emphasis or exaggeration. This helps in building up the auditory pattern, the visual association, and in directing the movement at the outset, preventing wrong speech patterns. First sentences may also be directed, as a whole, so that it becomes easy for the child to say "I want my mama," and avoiding the common misuse of the pronoun "Me want."

The child may be taught to talk best in the infancy period, from about the 12th month to two and a half years and in such a way that his speech development is often superior from that time on. You will observe several such children in the film to be shown presently.

We find it to be a surer and safer method to teach children to talk correctly, to prevent the persistence of wrong impressions of sounds, and to prevent difficulties such as stuttering or unrhythmic speech which come about when the speech is not normally timed or there are conflicting patterns, or other incorrect learning features. Nature permits us to gain certain results at certain stages in development. Only adults know how difficult it is to acquire language when we are full-grown. We believe that much more work should be done to rescue speech defectives of the pre-school age, and that we should be making more studies of children who are acquiring the wrong habits of speech at the outset. The best time to begin training in such cases we find, is between the 12th and the 30th month. Furthermore, this precept is in line with some of the most recent experimentation with young children in fields closely allied to speech, where nature begins to initiate the finer accessory muscles, after she has first established the basic use of the larger fundamental muscles. We may all teach children to talk, if we take the time and patience to acquire a somewhat different method and type of training.

Among other matters, a research committee for the School is undertaking studies of the beginnings of stuttering, verbatim recordings of children before and after speech training has been started, motion picture studies of various phases of speech development and presentation of methods by means of the films.

Dr. Lee Edward Travis, who has recently joined the staff at the University of Southern California, describes research projects on which he is working at the school as follows: one involves a study of the relative effectiveness of auditory, visual, kinaesthetic and verbal factors in learning, and of individual differences in the use of these factors in the acquisition of new skills and units of behavior. The

other project involves a comparative study of the behavior, particularly of the expressive components, of delayed speech and normal speaking children.

Summarizing we may say (1) It is possible to teach the child to talk, by strengthening the bonds between the more easily aroused motor and kinaesthetic centers of the brain, in some children, with the visual and auditory perceptual areas, until finally patterns are built up which involve gaining a knowledge of the place where movement begins, the form of the movement and finally the timing of the movement.

(2) This is possible not only with normal children who are slow in talking, but may be applied to the baby learning to talk, and may act as a preventive measure to rule out inaccuracies as they occur.

(3) It is a new therapy for the use with specially handicapped children such as the deafened or hard-of-hearing child, and the child with visual defects or loss of vision so that he must depend on moto-kinaesthetic and auditory cues very largely.

(4) It is a possible preventative for stuttering, when used with young children and many stubborn cases even of the adult level have been successfully trained into normal speech habits by this therapy.

(5) It opens up a new field for the worker with such handicapped children as birth injury cases, meningitis, infantile paralysis and encephalitis.

(6) It increases the facility with which the mentally deficient child may acquire language and aids in the substitution of normal habits to replace peculiarities and mannerisms, by giving the child a new skill with finer accessory muscles, where he has previously been able to employ only larger fundamental muscles chiefly.

(7a) It solves a problem which has puzzled many teachers and parents, as well as physicians and psychologists for centuries, by finding a way out for the imprisoned mind of a child who must necessarily lead a shut-in, narrowed existence, unless he finds a release through verbalization, and establishes a new environment for himself, by means of that most important of social tools, articulate speech, necessary for the exchange of ideas. Lacking speech, he is forever isolated to a large extent from his kind, and much restricted as to possibilities.

(7b) The question often arises as to whether or not this direct approach does not make the child self-conscious. Our answer to this is that the child is conscious about the difference between his speech and normal speech and this causes peculiar behavior. After he has learned what to do and how to do it, he is actually free from self-consciousness in relation to speech and often is no longer to be considered a "behavior problem."

SOCIETY OF PROGRESSIVE ORAL ADVOCATES

The Twenty-second Annual Meeting of the Society of Progressive Oral Advocates was held at St. Louis, February 17–18–19, 1939, at Central Institute for the Deaf, with an attendance of 100 members and 36 visitors, representing 15 states and 27 schools.

The following program was presented:

MORNING SESSION—FRIDAY, FEBRUARY 17, 1939

Address of Welcome—Col. Edwin B. Meissner, Pres., Central Institute for the Deaf.

President's Address—Max A. Goldstein, M. D., LL. D.

Curriculum Revision at the Virginia School—Forest E. Finney, Prin., Virginia State School.

A Deaf Child's Diary of Every-day Living—Mary W. Robinson, Calif. School for the Deaf.

A Successful Experiment in Grading—Sarah E. Lewis, School for Deaf, Sioux Falls, S. D.

Demonstration by Teachers of Faculty of Central Institute for the Deaf—Preschool Orchestra by 20 Preschool Children, directed by Miss May Templeman.

Nursery School Work—Jane Y. Olmsted and Frances D. Maritz.

First Grade Class—May Templeman.

Demonstration in Reading—Rachel D. Davies.

Reading Activity on Books of Today and Yesterday—Jessie G. Skinner.

One Phase of an Enriched Curriculum in History—Alfred Thea.

AFTERNOON SESSION—FRIDAY, FEBRUARY 17, 1939

Social Education for the Deaf—Sister M. Maura, St. Mary's School for Deaf, Buffalo.

Lip-Reading for Children—Elizabeth H. Nitchie, Central Inst. for the Deaf.

A Demonstration of Class Instruction, Direction of Elizabeth H. Nitchie and Betty Euwer.

Operation of Recently Organized Deaf Prevention Clinic—Participated in by Otologists, Stenographers, Registration Clerk, Audiometrists, Nurse, and 25 members of the Senior Class of the Teachers Training College. Systematic examination and recording of a group of 35 children from St. Louis County Schools, previously examined by 4-A Group Audiometer.

A Project in Grade 2-B, "Learning Our Family Relationships"—Mayme Voorhees, New York School for Deaf, White Plains. (Read by Amelia E. Berry.)

The Natural Language Class—Hilda J. Liff, Lexington School for the Deaf, New York City.

MORNING SESSION—SATURDAY, FEBRUARY 18, 1939

Diagnostic Procedure for Incoming Pupils in the Corrective Speech Clinic—Mildred A. McGinnis, Supervisor, Speech Correction Dept., Central Institute for the Deaf.

The Speech Clinic of C. I. D. in Active Operation—Mildred A. McGinnis, Helen Fagan, Staff of Speech Department. Twenty Student-Teachers demonstrating 60 speech defectives with various types of defect, each pupil receiving an individual twenty-minute lesson.

The Acoustic Method. The various steps of the Acoustic Method were presented in sequence by Audrey Hicks, Lillian D. Smalley, Catherine L. Fogle, Sara Hill Silverman, Mary Frances McCall and Augusta Roeder. Announcement was made by Dr. Max A. Goldstein that the entire Oral School of C. I. D. would now function by application exclusively of the Acoustic Method. All classrooms have been equipped with group microphones and individual ear sets and each teacher is now applying the Acoustic Method to her class and adapting all approach by Oral Training to this Method.

(*Continued on page 192*)

SPEECH, VOICE, AND HEARING BIBLIOGRAPHY

(Continued)

1933–1938, Sept.

Submitted by

JEANNETTE DEAN-THROCKMORTON, M. D.,
Iowa State Medical Library, Des Moines, Iowa

APHASIA

SINGER, H. D., and LOW, A. A. *Acalculia (Henschen); Clinical Study.* Arch. Neurol. & Psychiat. 29: 467–498, March, 1933.

DIMITRI, V. *Aphasia Caused by Hemorrhage of Lenticular Zone; Clinical and Anatomic Study of Case.* Rev. Asoc. Med. Argent. 47: 1998–2002, Jan., 1933.

DIMITRI, V. *Clinical and Anatomic Studies of Eight Cases of Aphasia.* Rev. Asoc. Med. Argent. 47: 2002–2018, Jan., 1933.

FROSCHELS, E. *Aphasia; Formation and Co-ordination of Old and New Theories.* Ztschr. f. d. ges. Neurol. & Psyciat. 143: 589–604, 1933.

LOTMAR, F. *Aphasia: Pathophysiology of Increased Difficulty in Finding Words.* Schweiz. Arch. f. Neruol. u. Psychiat. 30: 86; 30: 322, 1933.

KETTER, K., and ZWIRNER, E. *Psychologic-Phonetic Study in Aphasia.* J. f. Psychol. f. Neurol. 44: 690–738, 1932.

MARINESCO, G., and KREINDLER, A. *Speech as Conditioned Reflex; Relation to Verbal Aphasia.* Ztschr. f. d. ges. Neurol. u. Psychiat. 145: 167–184, 1933.

GIRARD, J., and GRIMAUD. *Aphasia, Auditory; Case of Word Deafness.* Gaz. d. hop. 105: 1736–1737, Nov. 23, 1932.

WEBER, F. P. *Congenital Deficiency of Conscious Auditory Perception of Words (Word Deafness); Case.* Proc. Roy. Soc. Med. 26: 509–510, March, 1933.

FOURNIER, J. C. M., CERVINO, J. M., and CASTIGLIONI, C. A. *Aphasia, Motor; Bilateral Transitory Hyperalgesia with Hyperalgesic Reflexes in Patient with Hemiplegia and Broca's Aphasia; Case.* Rev. Neurol. 2: 693–698, Dec., 1932.

SINGER, H. D., and LOW, A. A. *Aphasia, Motor; Brain in Case in Which Improvement Occurred with Training.* Arch. Neurol. & Psychiat. 29: 162–165, Jan., 1933.

VAN GEHUCHTEN, P. *Aphasia, Motor; four Cases.* Bull. Acad. Roy. de Med. de Belgique 12: 525–526, Sept. 24, 1932.

BRUMNER, H., and STENGEL, E. *Aphasia, Motor; In Child with Sinistrolateral Otogenic Abscess of Temporal Lobe.* Ztschr. f. d. ges. Neurol. u. Psychiat. 142: 430–450, 1932.

SCHACHTER, M. *Aphasia, Sensory; Crossed Aphasia; Case.* Progres Med., 357–358, Feb. 25, 1933.

SCHLESINGER, B. *Aphasia, Visual; Pathologic Study of Left and Right Disturbance, Constructive Apraxia and Pure Agraphia; Case.* Monatschr. f. Psychiat. u. Neurol. 84: 238–242, Nov. 1932. (Comment on Zutt's article); reply by Zutt 84: 242–244, Nov., 1932.

MAYER, L. L. *Congenital Reading Disability; Strephosymbolia; Five Cases of Partial Word Blindness.* J. A. M. A. 100: 1152–1155, April 15, 1933.

LAST, S. L. *Psychopathologic Analysis of Optic-Agnostic Disturbances.* Arch. f. Psychiat. 99: 197–232, 1933.

BOREL. *Pure Traumatic Word Blindness; Psychology of Patients with Alexia from Time of Charcot and Dejerine to Present Time.* Bull. et mem. Soc. franc. d'opht. 45: 417–437, 1932.

GOLDSTEIN, K. *Analysis of Aphasia and Study of Fundamentals of Language.* J. de psychol. norm. et path. 30: 430–496, Jan.-Apr., 1933.

THIELE, R. *Aphasia, Apraxia and Agnosia.* Fortschr. d. Neurol., Psychiat. 5: 233–247, June, 1933.

FORSTER, E. *Aphasic Disturbances of Ability to Speak and of Ability to Understand Speech.* Med. Klin. 29: 1331–1333, Oct. 1, 1933.

RAWAK, F. *Clinical Aspects of Agraphia.* Arch. f. Psychiat. 99: 773–803, 1933.

DIMITRI, V. *Aphasia Due to Hemorrhage of Lenticular Zone; Clinical and Anatomical Study of Case.* Arch. argent. de Neurol. 8: 1–17, Feb., 1933.

CAHANE, M., and VASILCA, V. *Hemiplegia and Aphasia of Diphtheric Origin; Pathogenesis of Encephalomalacia; Case.* Cluj. med. 14: 347–349, 1933.

BELTRAN, J. R. *Logoplegia, Case.* Rev. Asoc. med. argent. 47: 2743–2749, June, 1933.

LEDOUX, E., and JACQUOT, G. *Mechanism of Hemiplegia and Aphasia in Patients with Arrhythmia; Eight Cases.* Presse med. 41: 1285–1286, 1933.

JELLINEK, A. *Phenomenology of Amusia; Expressive Amusia and Aphasia of One Who Sings to Accompaniment of Stringed Instrument.* Jahrb. f. Psychiat. u. Neurol. 50: 115–141, 1933.

ROUQUIER, A. *Psychophysiologic Mechanism of Pithiatric Aphasia; Functional Origin Independent of Suggestion; Case.* Encephale 28: 294–301, April, 1933.

SCHAFFER, K. *Relation of Amnesic Aphasia to Kurt Goldstein's "Categorical States."* Arch. f. Psychiat. 100: 427–431, 1933.

MAYENDORF, M. V. *So-called Parietal Aphasia.* Ztschr. f. d. ges. Neurol. u. Psychiat. 147: 1–49, 1933.

WEBER, F. P. *Congenital Deficiency of Conscious Auditory Perception of Words (Word Deafness).* J. Neurol. & Psychopath. 13: 344–348, April, 1933.

NAVARRO, J. C., and BLACK, J. A. *Aphasia, Motor; Anarthria (Ataxic Aphasia) in Child; Etiopathogenesis and Therapy.* Arch. argent. de pediat. 4: 397–403, June, 1933.

ROUSSY, G., and LEVY, G. *Aphasia, Motor; Curious Case of Aphasia Resulting from Encephalopathy (Meningitis) in Infancy; Comparison with Two Other Cases Due to Encephalopathy in Infancy.* Vol. jubiliere, Marinesco, 579–598, 1933.

GRIGORESCU, D., and DIMITRIU, A. *Motor Aphasia; Anarthria Due to Traumatic Cortical Lesion.* Spitalul 53: 305–309, July-Aug., 1933.

JACQUET, P., and LEBLANC, M. *Pure Motor Aphasia; Case.* Bull. et mem. Soc. med. d. hop. de Paris. 49: 835–839, June 26, 1933.

ENGERTH, G., and URBAN, H. *Aphasia, Sensory; Disturbance of Artistic Ability in Aphasia.* Ztschr. f. d. ges. Neurol. u. Psychiat. 45: 753–787, 1933.

BENDER, L. *Disturbances in Visuomotor Gestalt Function in Organic Brain Disease Associated with Sensory Aphasia.* Arch. Neurol. & Psychiat. 30: 514–537, Sept., 1933.

POTZI, O. *Polyopia and Agnosia; Study in Pathophysiology of Visual Disturbances Due to Cerebral Conditions.* Jahrb. f. Psychiat. u. Neurol. 50: 57–77, 1933.

GILL, J. M. *Aphasia; Consideration of Views of John Hughlings Jackson and Henry Head.* M. J. Australia 1: 122–125, Jan. 27, 1934.

WEISENBURG, T. H. *Aphasia; Charles K. Mills Memorial Lecture.* Arch. Neurol. & Psychiat. 31: 1–33, Jan., 1934.

STENGEL, E. *Conduction Aphasia; Case.* Ztschr. f. d. ges. Neurol. u. Psychiat. 149: 266–291, 1933.

VON STOCKERT, F. G. *Gerstmann Syndrone (Finger Agnosia) in Connection with Disturbances of Speech and of Ability to Write (form of Aphasia).* Monatschr. f. Psychiat. u. Neurol. 88: 121–151, March, 1934.

GALPERIN, P. Y., and GOLUBOVA, R. A. *Mechanism of Paraphasia of Complex Type.* Sovet. psikhonevrol, No. 6, 38–48, 1933.

STONE, L. *Paradoxical Symptoms (Visual Hallucinations, Aphasia, etc.) in Right Temporal Lobe Tumor; Case.* J. Nerv. & Ment. Dis. 79: 1–13, Jan., 1934.

ZUCKER, K. *Pathologic Functions in Amnesic Aphasia.* Monatschr. f. Psychiat. u. Neurol. 87: 65, Nov.; 193, Dec., 1933.

WAGNER, W., and MAYER, K. *Psychologic Study of Aphasic Speech Disturbance in Girl 12 Years Old.* Monatschr. f. Psychiat. und Neurol. 87: 108–155, Nov., 1933.

ORTON, S. T. *Aphasia; Studies in Language Function.* A. Research Nerv. & Ment. Dis., Proc. 13: 614–632, 1932.

ODOBESCO, G., and VASILESCO, H. *Subcortical Aphasia; Motor and Total Aphasia; Two Cases.* Semaine d. hop. de Paris. 10: 123–124, Feb. 28, 1934.

STENGEL, E., and ZELMANOWICZ, J. *Aphasia, Motor; Polyglot Aphasia; Case.* Ztschr. f. d. ges. Neurol. u. Psychiat. 149: 292–311, 1933.

LEBEDINSKY, M. S. *Aphasia, Motor; Psychologic Analysis of Case.* Sovet. psikhonevrol., No. 6: 49–58, 1933.

SOBOL, M. E. *Relation Between Aphasic and General Motor Disturbances.* Sovet. psikhonevrol., No. 5: 45–51, 1933.

TKACHEV, R. A. *Aphasia, Visual; Congental Alexia.* Sovet. nevro-pat., psikhiat. i. psikhogig. No. 10, 2: 83–91, 1933.

CHAMBERS, E. R. *Congenital Word-blindness.* Bristol Med.-Chir. J. 51: 41–46, 1934.

ZUCKER, K. *Analysis of Disturbed Function in Aphasia.* Brain 57: 109–127, June, 1934.

GOLDSTEIN, K. *Critical and Factual Study of Basic Problems in Psychopathology, Particularly in Realm of Aphasia.* Schweiz. Arch. f. Neurol. u. Psychiat. 34: 69–93, 1934.

BIZARRI, A. *Psychologic Principles in Theories Concerning Aphasia.* Arch. di pat. e. clin. med. 13: 569–606, May, 1934.

ROGERSON, C. H. *Congenital Auditory Imperception (Word Deafness).* Guy's Hosp. Repts. 84: 237–249, April, 1934.

GROSSI, F., and FAMIGLIETTI, M. *Purely Toxic Motor Aphasia Following Pneumonia; Case in Infant.* Pediatria 42: 874–878, July, 1934.

AMYOT, R., and SAINT-GERMAINE. *Case of Right Hemiplegia and Sensorimotor Aphasia; Etiologic Study.* Union med. du Canada. 63: 1105–1114, 1934.

FROMENT, J., and FEYEUX, A. *Pure Motor Aphasia; Simulated Anarthria Re-educated by Recalling Sounds to Memory; Case.* Rev. Neurol. 1: 1058–1066, June, 1934.

CHANDRA, K. *Aphasia.* Indian M. Rec. 55: 1–2, Jan., 1935.

PAULIAN, D. E., and CARDAS, M. *Aphasia After Carbon Monoxide Poisoning in Patient With Syphilitic Arteritis.* Romania med. 13: 89, April 1, 1935.

BARUK, H., and POUMEAU-DELILLE. *Cataleptic Attacks with Aphasia, Apraxia, and Disturbances of Instinct; Death in Cataleptic State; Case.* Rev. Neurol. 2: 833–839, Dec., 1934.

TZANCK, A., and SCHIFF, P. *Aphasia Following Administration of Tetanus Antiserum in Syphilitic; Case Resembling General Paralysis.* Bull. et mem. Soc. med. d. hop. de Paris. 51: 536–538, April 1, 1935.

WEISENBURG, T. H. *Aphasia in Brain Tumors.* Ann. Surg. 101: 373–382, Jan., 1935.

BELTRAN, J. R. *Aphasia; Logoplesia; Case.* Arch. argent. de psicol. norm., pat., terap. neuro ment. y cien. afines. 1: 13–17, July, 1933.

DAWSON, S. *Psychology and Aphasia.* Post-Grad. M. J. 11: 139–144, Apr., 1935.

FERRE, L. B., and PAUSAS, J. T. *Transitory Aphasia in Patients with Arrhythmia.* Ann. de Hosp. de Santa Creu i Sant Pau. 8: 184–185, May 15, 1934.

BIOLATO, D. *Transitory Aphasia in Persons Injured in Left Temporoparietal Region without Cranial Lesions; Two Cases.* Bull. e mem. Soc. piemontese di chir. 4: 1746–1760, 1934.

GLASER, E. M. *Treatment of Agraphia by Kinesthetic Stimulation and Psychotherapy.* J. Nerv. & Ment. Dis. 81: 47–54, Jan., 1935.

STEIN, L. *Treatment of Associative Aphasia.* Monatschr. f. Ohrenh. 68: 920–935, Aug., 1934.

RATNER, R. G. *Unusual Case of Optic and Autotopic Agnosia.* Monatschr. f. Psychiat. u. Neurol. 89: 349–364, Oct., 1934.

KIRSCHNER, J. *Pure Word Deafness Combined with Inability to Understand What Is Read; Case.* Ztschr. f. d. ges. Neurol. u. Psychiat. 151: 457–464, 1934.

VICTORIA, M. *Aphasia, Motor; Irrefutable Arguments Against Theory that Aphasia is Due to Lesions of Left Third Frontal Convolution; Report of Case and Review of Literature.* Semana med. 2: 1771–1777, Dec. 6, 1934.

MARINESCO, G., and GRIGORESCO, D. *Aphasia, Sensory; Crossed Aphasia, with Hemiplegia; Case. Rev. neurol.* 63: 291–295, Feb., 1935.

FEUCHTWANGER. *Optic-constructive Agnosis; Pathology of Optic Power of Conception.* Ztschr. f. d. ges. Neurol. u. Psychiat. 151: 469–496, 1934.

DUGUID, K. *Congenital Word-blindness and Reading Disability.* Guy's Hosp. Rep. 85: 76–93, Jan., 1935.

VERMEYLEN, G. *Aphasia; Agnoso-apraxic Encephalosis* (Pick's Disease); *Case.* J. belge de neurol. et de psychiat. 35: 453–455, Aug. 1935.

PRANDI, G. *Aphasia Appearing in Typhoid and Lobar Pneumonia; Cases in Children.* Pediatria d. med. prat. 10: 240–245, April, 1935.

OKA, M. G. *Caisson Sickness Presenting Aphasia As One of Chief Symptoms; Two Cases.* Indian M. Gaz. 70: 629–630, Nov., 1935.

FERRANINNI, L. *Case of Aphasia with Paralysis of Right Arm and Slight Paralysis of Right Leg and Face Due to Softening of Cerebral Cortex.* Minerva med. 1: 649–652, May 10, 1935.

PFERSDORFF, C. *Classification of Aphasic Language; Relation to Schizophrenic Dissociation.* Ann. med.-psychol. (pt. 2) 93: 1–11, June, 1935.

RADERMECKER. *Encephalitis with Aphasia in Child 12 Years Old; Hereditary Syphilis As Cause.* Arch. med. belges 88: 165–175, July, 1935.

MOLINA, T. *Hemianopia with Temporary Aphasia; Case.* Arch. de oftal. de Buenos Aires 10: 552–557, July, 1935.

DUVOIR, M., PERISSON, J., and BERTRAND, I. *Left Hemiplegia in Right-handed Person, Accompanied by Clinical Signs of Aphasia with Predominance of Agraphia; Softening of Corpus Callosum.* Bull. et. mem. Soc. med. d. hop. de Paris 51: 1071–1080, July 1, 1935.

BINSWANGER, L. *Present Status of Knowledge of Aphasia.* Schweiz. Arch. f. Neurol. u. Psychiat. 36: 52–57, 1935.

FLORENSKY, J. A. *Logopedia in Aphasia; "Mirror Inversion" in Origin of Paraphasia.* Riv. de neurol. 8: 405–438, Aug., 1935.

CURRAN, F. J., and SCHILDER, P. *Paraphasic Signs in Diffuse Lesions of Brain.* J. Nerv. & Ment. Dis. 82: 613–636, Dec., 1935.

THIELE, R. *Aphasia; Review of Literature.* Fortschr. de Neurol., Psychiat. 7: 255–270, July, 1935.

MOSCHINI, S. *Right Hemiplegia and Aphasia After Scarlet Fever in Child Five Years Old.* Pediatria d. med. prat. 10: 408–420, July, 1935.

REPETTO, R. L., and COURTIS, B. *Right Homonymous Hemianopia with Speech Disturbances (Amnesic Aphasia); Case.* Rev. Asoc. med. argent. 49: 829–833, June, 1935.

JELLINEK, A. *Aphasia; Significance of Physiognomical Experience.* Monatschr. f. Ohrenh. 69: 219–224, Feb., 1935.

PANARA, C. *Aphasia, Motor; Following Paratyphoid B; Case.* Gior. di med. mil. 83: 569–573, June, 1935.

THOMAS, A., and SORREL, E. *Aphasia, Motor; Caused by Cranial Fracture and Brain Injury; Case in Child.* Rev. neurol. 63: 893–896, June, 1935.

MALAMUD, N. *Aphasia, Sensory; Case.* Univ. Hosp. Bull., Ann Arbor 1: 53, Dec., 1935.

SASAKI, T. *Aphasia, Visual; Alexia, Case.* Nagasaki Igakkwai Zassi 13: 710–711, May 25, 1935.

VAMPRE, E. *Disturbances in Reading of Cerebral Origin; Five Cases.* Arq. Inst. Penido Burnier. 3: 125–150, Dec., 1934.

WOLF, I. J. *Strephosymbolia vs. Congenital Word Blindness.* Proc. Am. A. Ment. Deficiency. 59: 446–465, 1935.

BADONNEL, and AJURIAGUERRA, J. *Aphaso-agnoso-apraxic Syndrome in Presenile Dementia; Case.* Ann. med. psychol. (pt. 2). 93: 798–805, Dec., 1935.

DEROM, E. *Cranial Fracture with Compression and Aphasia Due to Area of Cerebral Destruction; Operation with Recovery in Child 19 Months Old.* J. de chir. et ann. Soc. belge de chir. 34: 577–581, Oct.-Nov., 1935.

SCHAECHTER, M. *Crossed Aphasia with Hemiplegia; Clinical Study of Case.* Rev. med. de la Suisse Rom. 55: 947–950, Dec. 25, 1935.

MUNCIE, M. *Fingeragnosis* (Gerstmann). Bull. Johns Hopkins Hosp. 57: 330–342, Dec., 1935.

VAN VALKENBERG, C. T. *Aphasia; in Child 4 Years Old.* Nederl. tijdschr. v. geneesk. 80: 827–834, Feb. 29, 1936.

GUIRAUD, P., and FERDIERE, G. *Aphasia in Syphilis and General Paralysis; Problems of Diagnosis and Therapy.* Ann. med.-psychol. (pt. 1). 94: 404–407, March, 1936.

HOFF, H., and POTZI, O. *Aphasia; New Parieto-occipital Syndrome; Ideomotor Visual Paralysis, Disturbance of Body Scheme (Lack of Awareness of Certain Portions of Body) and Absence of Central Portion of Visual Field.* Jahrb. f. Neurol. u. Psychiat. 52: 173–218, 1935.

MELLEN, H. S., and POLOZKER, I. L. *Nominal or Amnesic Aphasia with Report of Case.* J. Michigan M. Soc. 35: 245–249, April, 1936.

LEBEDINSKY, M. S. *Aphasia; Peculiarities of Thinking in Patients.* Wien. klin. Wchnschr. 49: 137, Jan. 31, 1936; 179, Feb. 7, 1936.

SKLYAR, N. I. *Psychosis as Aphasia and So-called Palinbiosis.* Sovet. psikhonevrol., No. 6: 89–93, 1935.

CONSTANESCU, M. N., and PETROSANU, I. N. *Right Hemiplegia and Aphasia Beginning with Epileptiform Crisis of Jackson Type After Spinal Anesthesia with Procaine Hydrochloride; Case.* Rev. de.chir., Bucuresti 38: 125–128, Sept.-Dec., 1935.

STENGEL, E. *Transcortical Aphasia.* Zeitschr. f. d. ges. Neurol. u. Psychiat. 154: 778–782, 1936.

MOREL, F. *Hearing in Sensory Aphasia with Discussion on Cerebral Localization of Auditory Function.* Encephale 30: 533–553, Sept.-Oct., 1935.

WETTERWALD, M. *Aphasia, Motor; Temporary Aphasia During Pregnancy; Two Cases.* Schweiz. med. Wchnschr. 66: 56–57, Jan. 11, 1936.

SOLOMOVICI, H. *Aphasia, Motor; Hysterical Aphasia Simulating Cerebral Abscess in Patient with Chronic Double Otitis Media Who Had Been Previously Operated for Left Labyrintho-meningitis.* Ann. d'oto-laryng. 63–66, Jan., 1936.

GEHUCHTEN, P. V. *Tuberculoma of Pons Varolli and of Cerebellum with Symptoms of Left Frontal Lobe Tumor; Postopeative Motor Aphasia of Broca's Type.* J. belge de neurol. et de psychiat. 36: 61–68, Feb., 1936.

FAURE-BEAULIEU. *Aphasia, Sensory; Wernicke's Aphasia Due to Grave Carbon Monoxide Poisoning; Case.* Presse med. 45: 81–82, Jan. 11, 1936.

BRANDER, T. *Congenital Word Blindness and Similar Disturbances During School Age.* Finska läk.-sällsk. handl. 77: 601–618, 1935.

BRANDER, T. *Congenital Word Blindness Following Birth Trauma.* Monatschr. f. Kinderh. 64: 55–61, 1935.

——— *Experiences of Sufferer from Word-blindness.* Brit. J. Ophth. 20: 73–76, Feb., 1936.

LEY, J., and TORDEUR, G. W. *Developmental Alexia and Agraphia in Uniovular Twins.* J. belge de neurol. et de psychiat. 36: 203–222, April, 1936.

FROMENT, J. *Aphasia; Diagnosis of Language Difficulties and Therapeutic Indications Derived from Diagnosis.* Paris med. 2: 237–250, Oct. 3, 1936.

MARENESCO, G., and SAGER, O. *Aphasia; Electro-encephalography.* Bull. Acad. de med. Paris. 116: 182–186, Oct. 6, 1936.

BAONVILLE, H., and CAHEN, J. *Meningioma of Base of Brain with Dementia and Aphasia As Predominating Syndrome.* J. belge de neurol. et de psychiat. 36: 602–611, Sept., 1936.

LUGONES, C. *Metastatic Cerebral Abscess in Otitis with Amnesic Aphasia; Case.* Semana med. 1: 1239–1249, April 16, 1936.

FILIMONOV, I. N. *Method for Study of Aphasic Disorders.* Sovet. psikhonevrol. n. 1: 17–30, 1936.

MARCONDES, D. *Pure Form of Developmental Agraphia; Case in Girl 10 Years Old.* Rev. neurol. e psychiat. de Sao Paulo 2: 40–43, Jan., 1936.

LOTMAR, F. *Recent Battles Over Conception of Aphasic Disturbances.* Schweiz. Arch. f. Neurol. u. Psychiat. 38: 97–149, 1936.

KENNEDY, F., and WOLF, A. *Relationship of Intellect to Speech Defect in Aphasic Patients, with Illustrative Cases.* J. Nerv. & Ment. Dis. 84: 125, Aug.; 293, Sept., 1936.

FANIELLE, G. *Sudden Aphasia Due to Cerebral Abscess Following Tooth Extraction; Two Cases.* Liege. med. 29: 521–524, May 3, 1936.

SHORT, A. R. *Traumatic Aphasia and Alexia.* J. Neurol. & Psychopath. 17: 74–76, July, 1936.

MINKOWSKI, M. *Types of Aphasia Peculiar to Linguists.* Schweiz. med. Wchnschr. 66: 697–699, July 18, 1936.

BOURGUIGNON, G., and DEJEAN, R. *Variations of Vestibular Chronaxia with Hemiplegia and Aphasia.* Compt. rend. Acad. d. sc. 203: 500–502, Aug., 1936.

BERZE, J. *Pseudo-aphasic Auditory Illusions in Schizophrenia.* Allg. Ztschr. f. Psychiat. 104: 302–335, 1936.

BARASCH, O. *Aphasia, Motor; Hystero-organic Aphasia; Case.* Rev. stiint. med. 25: 697–700, May, 1936.

NOTTI, H. J. *Aphasia, Motor; Case with Right Hemiplegia in Convalescent Period of Diphtheria; Case.* Semana med. 1: 1392–1394, April 30, 1936.

MARANI, B. D. *Aphasia, Motor; Due to Brain Edema and Cerebral Chloridemia; Case.* Policlinico (sez. prat.) 43: 1522–1528, Aug. 24, 1936.

BALDI, F. *Singing of Patients with Aphasia; Possible Relation to Conditioned Reflexes; Application of Theory in Speech Re-education of Aphasic Persons.* Riv. di neurol. 9: 217–232, June, 1936.

HEERNU, J. *Wernicke's Aphasia in Syphilitic.* J. belge de neurol. et de psychiat. 36: 397–404, June, 1936.

RONNE, H. *Clinical Testing of School Children's Reading Abilities (Tests for Alexia).* Acta ophth. 14: 39–56, 1936.

LAUBANTHAL, F. *"Congenital Word Blindness"; Clinical Study of Irs Relation to Forms of Partial Feeblemindedness and Their Heredity.* Ztschr. f. d. Neurol. u. Psychiat. 156: 329–360, 1936.

GJESSING, H. G. A. *Transitory Word Blindness Associated with Right Homonymous Hemianopia; Case in Patient with Cancer of Prostate Gland.* Arch. Ophth. 16: 5–7, July, 1936.

THIELE, R. *Aphasia.* Fortschr. d. Neurol., Psychiat. 9: 81–92, Feb., 1937.

TRILLAT, P., and MAGNIN, P. *Aphasia and Right Hemiplegia at Onset of Labor at 7½ Months; Case.* Lyon med. 159: 173–178, Feb. 14, 1937.

MARINESCO, G., SAGER, O., and KREINDLER, A. *Electro-encephalographic Studies in Aphasia; Phenomena of Restitution and Compensation in Lesions of Brain.* Bull. Acad. de med. Paris. 116: 383–385, Nov. 17, 1936.

SELETSKIV, V. V. *Aphasia; Fingeragnosia and Acalculia.* Sovet. psikhonevrol. No. 10: 82–87, 1936.

ENGERTH, G. *Phonophysiognomic Observations in Retrogressive Stage of Aphasic Disturbances.* Wien. klin. Wchnschr. 50: 342–344, March 5, 1937.

TORP, H. *Aphasia, Problems.* Nord. med. tidskr. 13: 441–446, March 20, 1937.

POTZI, O., and STENGEL, E. *Syndrome Consisting of Combination of Conduction Aphasia and Pain Asymbolia.* Jahrb. f. Psychiat. u. Neurol. 53: 174–207, 1936.

CHESHER, E. C. *Aphasia; Technic of Clinical Examinations.* Bull. Neurol. Inst. New York. 6: 134–144, Jan., 1937.

KAUDERS, O. *Aphasia, Auditory; Isolated Sensory Amusia and Partial Agnosia; Case.* Jahrb. f. Psychiat. u. Neurol. 54: 119–132, 1937.

HOFF, H., and POTZI, O. *Anatomic Examination of Patient with Instrumental Amusia Type of Motor Aphasia Due to Cerebral Glioma.* Jahrb. f. Psychiat. u. Neurol. 54: 89–118, 1937.

VICTORIA, M. *Lesion of Third Left Frontal Convolution Without Aphasia.* Encephale 1: 85–92, Feb., 1937.

NIELSEN, J. M. *Possibility of Pure Motor Aphasia.* Bull. Los Angeles Neurol. Soc. 1: 11–14, March, 1936.

VEIDERS, E. *Aphasia, Visual; Study of Agnosias, Alexia and Disorders of Orientation.* Arch. f. Psychiat. 105: 433–474, 1936.

HUGHES, C. A. *Aphasia, Visual; Congenital Dyslexia.* Tr. Ophth. Soc. U. Kingdom 56: 307–311, 1936.

RONNE, H. *Congenital Word Blindness in School Children.* Tr. Ophth. Soc. U. Kingdom 56: 311–333, 1936.

INGHAM, S. D., and NIELSEN, J. M. *Interpretation Dissociated from Recognition of Visual Verbal Symbols Illustrated by Case of Complete Major (Left) Temporal Lobectomy.* Bull. Los Angeles Neurol. Soc. 2: 1–10, March, 1937; abstr., Tr. Am. Neurol. A. 62: 33–36, 1936.

HOFF, H., and POTZI, O. *Unusual Syndrome (Optic Simultaneous Agnosis Changing Into Stupor) in Patient with Tumor of Mesencephalon.* Jahrb. f. Psychiat. u. Neurol. 54: 13–54, 1937.

FRANK, H. *"Word Blindness" in School Children; Predominant Causal Role of Delayed Dvelopment of Perceptional Functions.* Tr. Ophth. Soc. U. Kingdom 56: 231–238, 1936.

HUBER, H. G. *Acute Idiopathic Meningeal Hemorrhage with Aphasia in Child 5 Years Old; Differential Diagnosis of Meningo-encephalitis.* Kinderärztl. Praxis 8: 414–419, Oct., 1937.

MARINESCU, G., and GRIGORESCU, D. *Anatomicoclinical Analysis of Case of Mixed Aphasia.* Spitalul 57: 183–191, May, 1937.

MANZI, L. *Hemiplegia and Aphasia of Postpartum Period; Case Following Delivery and Case Following Incomplete Abortion.* Arch. di ostet. e ginec. 1: 101–111, March-April, 1937.

STENGEL, E. *Relation Between Mental Disorder and Speech Defects; Theories of Verbal Aphasia and Paraphasia.* Monatschr. f. Psychait. u. Neurol. 95: 129–173, April, 1937.

DOZIER, P. *Etiologic and Clinical Types of So-called Nerve Deafness from Affections of Brain; Organic Factors; Congenital Word Deafness.* Laryngoscope 47: 516–519, Aug., 1937.

CHESHER, E. C. *Etiologic and Clinical Types of So-called Nerve Deafness; from Affections of Brain; Organic Factors; Acquired Word Deafness (in Relation to Brain Tumors).* Laryngoscope 47: 520–526, Aug., 1937.

TAILLENS. *Aphasia, Motor; in Children; Three Cases.* Schweiz. med. Wchnschr. 67: 988–989, Oct. 9, 1937.

VIE, I. M. *Serious Speech Disturbances in Intelligent Child with Motor Debility.* Monatschr. f. Ohrenh. 71: 1075–1076, Sept., 1937.

BECK, E. *Aphasia, Sensory.* Ztschr. f. d. ges. Neurol. u. Psychiat. 158: 193–203, 1937.

PALOMBA, G. *Development of Pure Alexia After Decompressive Operation for Brain Cyst with Sensory Aphasia; Case.* Riforma med. 53: 1445–1451, Oct. 9, 1937.

MARCHAND, L. *Post-traumatic Aphasia and Epilepsy; Meningocortical Cicatrices of Left Temporo-occipital Region Found at Autopsy; Case.* Ann. med.-psychol. (pt. 1) 95: 768–771, May, 1937.

LAUBENTHAL, F. *Siblings with Congenital Word Blindness.* Ztschr. f. d. ges. Neurol. u. Psychiat. 158: 321–324, 1937.

STAHL, R. *Congenital Word Blindness: Case.* Monatschr. f. Psychiat. u. Neurol. 97: 1–13, Sept., 1937.

HOFF, H., and POTZI, O. *True Word Blindness in Patient with Brain Tumor; Case.* Nervenarzt 10: 385–395, 1937.

PALOMBA, G. *Development of Pure Alexia After Decompressive Operation for Brain Cyst with Sensory Aphasia; Case.* Riforma med. 53: 1445–1451, Oct. 9, 1937.

HOFF, H., and POTZI, O. *Optic-agnostic Disturbance of Memory for Faces; Relation to Retrogression of Word Blindness.* Ztschr. f. d. ges. Neurol. u. Psychiat. 159: 367–395, 1937.

HANSEN, P. A. S. *Word Blindness in Children.* Ugesk. f. laeger 99: 520–522, May 13, 1937.

PFERSDORFF, C. *Amnesic Aphasia Following Cerebral Trauma; Case.* Strasbourg med. 97: 635–636, Dec. 15, 1937.

GEORGESCO, P., and PODEANU, G. *Cranial Trauma Followed by Aphasia; Case.* Rev. de chir., Bucuresti 40: 193–195, Jan.-Feb., 1937.

LINDQVIST, T. *Disorders of Intellect in Aphasia and Similar Conditions.* Acta psychiat. et neurol. 12: 411–427, 1937.

AUSTREGESILO. *Aphasia Due to Disturbance of Left Parietal Lobe.* Rev. de psiquiat. y criminol. 2: 433–446, July-Aug., 1937.

BOGUSH, N. R. *Epileptic Equivalents to Form of Aphasic Attacks.* Sovet. psikhonevrol., No. 1: 76–79, 1937.

NIELSEN, J. M. *Gerstmann Syndrome; Finger Agnosia, Agraphia, Confusion of Right and Left and Acalculia; Comparison of this Syndrome with Disturbance of Body Scheme Resulting from Lesions of Right Side of Brain.* Arch. Neurol. & Psychist. 39: 536–560, March, 1938.

MARCUS, H. *Aphasia; Localization of Agraphia.* Acta psychiat. et neurol. 12: 431–446, 1937.

BOSSELMAN, and KRAINES, S. H. *Mental Changes Including Aphasia in Patient with Sickle Cell Anemia.* Am. J. Psychiat. 94: 709–712, Nov., 1937.

TORP, H. *Problems of Aphasia.* Acta psychiat. et neurol. 12: 397–410, 1937.

CONSTANESCU, PETROSANU, and FRANK. *Right Hemiplegia and Aphasia with Initial Epileptiform Seizures of Jacksonian Type, Following Lumbar Anesthesia with Procaine Hydrochloride.* Zentralbl. f. Chir. 65: 83–85, Jan. 8, 1938.

CRITCHLEY, M. *Aphasia; Testamentary Competency of Patient.* Practitioner 140: 189–196, Feb., 1938.

TESTA, M. *Two Cases of Porencephaly of Left Cerebral Hemisphere; Contribution to Study of Aphasia.* Rassegna di fisiopat. clin. e terap. 10: 1–41, Jan., 1938.

USTVEDT, J. *Aphasia, Motor; Musical Functions in Patients with Aphasia.* Acta med. Scandinav., supp. 86: 1–737, 1937.

CURRIER, F. P. *Certain Reading Disabilities (in Left and Right-handedness) As Related to Speech.* J. Michigan M. Soc. 37: 414–418, May, 1938.

ROTTGEN, P., SELBACH, H., STOCKERT, V., and TONNIS, W. *Development of Transitory Focal Symptoms (Motor Aphasia), Psychic Ditsurbances and Changes in Cerebro-spinal Fluid Following Operations on Brain.* Zentralbl. f. Neurochir. 3: 12–46, 1938.

SOLOMOVICI. *Hysterical Cerebral Manifestations (Aphasia) as Indication for Mastoid Intervention in Chronic Suppurative Otitis Media; Results in Case.* Ann. d'oto-laryng., 36–40, Jan., 1938.

PAULIAN, D. *Physiopathology of Aphasic Disorders Due to Softening or Abscess of Left Frontal Lobe.* Rev. san. mil., Bucuresti 36: 317–325, April, 1937.

MARINESCO, GRIGORESCO, and AXENTE, S. *Crossed Aphasia; Wernicke's Aphasia with Left Hemi-anopia and Hemiplegia in Right-handed Patient; Further Report of Case.* Encephale 1: 27–46, Jan., 1938.

MARINESCO, SAGER, and KREINDLER. *Electro-encephalogram in Certain Pathologic Conditions, with Special Reference to Wernicke's Aphasia, Cerebral Tumors and Coma.* Presse med. 46: 650–654, April 27, 1938.

CERVELLO. *Wernicke's Aphasia; Clinical and Anatomicopathologic Study of Case.* Cervello 16: 345–360, Nov. 15, 1937.

CARR, T. E. A. *Congenital Word-blindness.* Tr. Ophth. Soc. U. Kingdom (pt. 2, 1937) 57: 579–590, 1938.

FRANCESCHETTI, and DE MORSIER, G. *Pure Alexia of Traumatic Origin with Hemianopia in Right Lower Quadrant; Case.* Rev. de oto-neuro-opht. 16: 125–127, Feb., 1938.

MODY, S. H. *Aphasia and Complete Right Hemiplegia After Scorpion Bite.* Indian M. Gaz., 73: 285, May, 1938.

CRITCHLEY, M. *"Aphasia" in Partial Deaf-mute.* Brain 61: 163–169, June, 1938.

GOTO, T. *Aphasia, Motor; Cure by Pneumo-encephalography; Case.* Oto-rhino-laryng. 11: 539, June, 1938.

KLEIN, R. *Speech Disorders in Aphasia Due to Tumors of Temporal Lobe.* Monatschr. f. Psychiat. u. Neurol. 98: 189–237, June-July, 1938.

MARSALET, P. D. *Aphasic Disorder Does Not Constitute Static Deficiency; Study of Incomplete Forms of Wernicke's Aphasia With and Without Anarthria.* Encephale 1: 123–131, March, 1938.

ANGYAL, L. V., and LORAND, B. *Drawing Difficulties of Autopagnostic-aphasic Patients.* Arch. f. Psychiat. 108: 493–516, 1938.

THIELE, R. *Congenital Word-blindness; Case.* Monatschr. f. Psychiat. u. Neurol. 99: 371–399, April, 1938.

SOCIETY OF PROGRESSIVE ORAL ADVOCATES

(*Continued from page 180*)

BUSINESS SESSION

The following officers were chosen for the ensuing year:

MAX A. GOLDSTEIN, St. Louis.....*President*
ALICE COBURN, Chicago.....*Vice-President*
TRUMAN L. INGLE, Fulton.....*Vice-President*
DAN T. CLOUD, Jacksonville.....*Vice-President*
ELIZABETH GORSUCH, St. Louis.....*Secretary-Treasurer*
MARGARET SCYSTER, Jacksonville.....*Asst. Secretary-Treasurer*
Executive Committee: JULIA M. CONNERY, St. Louis, *Chairman;* ELIZABETH SCOTT, Chicago; LULU M. BRUCE, Danville; OLIVE RODGERS, Denver; EVELYN HUMPHREYS, Dayton.

A special committee appointed to consider changing the name of this organization reported through its chairman, JESSIE G. SKINNER, and submitted the name:

National Forum on Deafness and Speech Pathology

After a short discussion this name was adopted and henceforth the Society of Progressive Oral Advocates will be known as the "National Forum on Deafness and Speech Pathology."

The selection of the time and place of the next meeting of this society was left to the Executive Committee.

A vote of thanks was extended to Supt. TRUMAN L. INGLE for his courtesies in publishing the proceedings of the 21st Annual Meeting.

AFTERNOON SESSION, SATURDAY, FEBRUARY 18, 1939

Visual Aids—SUSAN CHRISTIAN, Indiana School for the Deaf.

The Effect of the Daily Item on Language—LULA M. BRUCE, Kentucky School for the Deaf.

Speech Without a Tongue—Demonstration of Two Patients—DR. E. LAURENCE KEYES, St. Louis.

Phonograph Reproduction of Speech by Man Without a Tongue. Movie Film, Unusual Activities of Muscles of the Tongue in Six Selected Cases—DR. MAX A. GOLDSTEIN.

Palate Setback Operation and Its Effect on Speech, with Lantern Demonstration and Presentation of Patients—DR. JAMES BARRETT BROWN, St. Louis.

The Acoustic Method in the Wisconsin School—MARLINE H. PARRISH, Wisconsin School for the Deaf.

BANQUET—HOTEL CHASE

AFTERNOON SESSION—SUNDAY, FEBRUARY 19, 1939

Residual Hearing and the Acoustic Method—DR. CORDIA C. BUNCH, Central Institute for the Deaf.

Testing for Speech Intelligibility in a Residential School for the Deaf—R. MOORE STELLE, Illinois School for the Deaf.

Language Development—ALLIE ARNOLD, Colorado School for the Deaf.

A Play "Colonial Days"—Pupils of C. I. D. under the Direction of the Senior Class of the Teachers Training College of C. I. D.

Tea—Tendered by the Faculty of Central Institute.

This report is offered for publication, including the program of the three-days' sessions and brief notes of explanation.

Elizabeth Gorsuch, *Secretary-Treasurer,*
National Forum on Deafness and Speech Pathology.